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The Halford Oration.[1]

ON SURGICAL EDUCATION.

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GEORGE BRITTON HALFORD was the first professor of the first medical school in Australia. When he arrived in Melbourne, in December, 1862, he was thirty-eight years old and had won a wide reputation by his original work in comparative anatomy and physiology. Indeed, it seems probable that, as Professor W. A. Osborne suggested in the first Halford Oration, had he remained in London instead of undertaking the difficult and arduous task of creating a new medical school in a remote corner of the world, he would have won enduring fame for his scientific work. But this was not to be. He elected to come to Melbourne, where he found three second-year medical students waiting to be taught, but no medical school in which to teach them. He at once set to work to draw up plans for the erection of the school, teaching the students meanwhile in a shed in the backyard of his private house. The medical school buildings were actually completed and ready for use in a little more than one year after Halford's arrival, a fact which demonstrates that, in those days, he must have been what is now described as a "live wire". But the creation of a medical school entails much more than the erection of buildings; it is necessary to devise a curriculum that will give its students an adequate

[1] Delivered at the Institute of Anatomy, Canberra, on November 23, 1936.

training, and it is essential to inspire in them a desire to profit from this training. Halford did both these things. He adopted Dr. Anthony Colling Brownless's suggestion that the curriculum should embrace a five-year period of study, one year more than that of the English medical schools of the time, and to his students he preached the doctrine of diligence in the pursuit of knowledge. The success of his efforts is shown by the calibre of the early graduates of the school, one of whom, later Professor Sir Harry Allan, was to succeed him in the task of developing the Melbourne medical school.

The spectacle of a professor, so over-burdened by teaching and administrative duties that little time is left to him for research, is by no means singular, and it therefore occasions no surprise to find that Halford's later researches lacked the brilliance of his earlier work. Halford made Melbourne and Melbourne marred Halford. Let us take comfort in the belief that he who builds a medical school upon a sound foundation confers a great benefit upon humanity and, as his school "grows in future fame", gains, in it, an increasingly impressive memorial. But Halford's memorial is not only the Melbourne medical school; it is also the high standard of medical education in this country, because, by enforcing a curriculum of what was then unparalleled severity, he established the tradition that our medical education should compare favourably with that obtainable elsewhere. This tradition has been sedulously fostered by his successors and, in paying a tribute to the founder of the school of which I am proud to be a graduate, I propose to show how Halford's principles have guided those responsible for the direction of post-graduate surgical education in Australasia.

It is perhaps not generally recognized that, during the actual lifetime of those who have today reached the allotted span of years, conditions of surgical practice have been revolutionized, and that, as a direct result, the problems of surgical education have become much more complex, and at the same time of much greater significance to humanity. A brief retrospect will show the truth of this statement. Two hundred years ago there was no surgical education worthy of the name in England. The craft of surgery had not been separated from the craft of barbery and the chief school for the surgeon of that time was the field of battle. There he could gain experience in amputating limbs, ligating blood vessels, and treating wounds, relatively simple operative procedures which embraced almost the whole field of the civil surgery of the day. Perhaps nothing more forcibly demonstrates the change in conditions of surgical practice between those days and the present time than the fact that there is no worse training for the modern surgeon than that given by the necessarily slapdash treatment of war casualties. Then, in the middle of the eighteenth century, came John Hunter, one of the greatest biologists and greatest surgeons of all time, whose work not only laid the foundation of the science of surgery, but also aroused greater interest in the problems of surgical education. At this time, the ancient alliance between the barbers and the surgeons was dissolved and the Company of Surgeons, the forerunner of the Royal College of Surgeons of England, was granted a charter.

The educational problems which confronted the new Company were not of great complexity, for surgical knowledge was limited, operations were comparatively rare, and candidates for the surgical profession were few in number. This is not surprising when we remember that operations in those pre-anæsthetic days could be performed successfully only by men who possessed unusual attributes of courage, skill and humanity. These were the days of the "master surgeons", as Professor F. Wood Jones calls them in his eloquent Syme Oration, for only supermen could hope to succeed when the patient was the conscious judge of the skill of his surgeon. There was, therefore, an apostolic succession in surgery, teaching was mainly by a system of apprenticeship and the mantle of the master surgeon fell naturally upon the shoulders of the most elect of his pupils. Successful surgeons had to possess technical skill of such high degree that the operation was over almost before the patient had time to feel the pain. It is true that there were some bad surgeons, for we read of one who struggled unsuccessfully for half an hour to remove a stone from the bladder, an operation performed by Cheselden in three minutes, and we have heard a story, possibly apochryphal, of a would-be dextrous operator who, with one swift sweep of the knife, amputated the leg of his patient, two fingers of his assistant, and the coat-tails of a spectator, but such men must soon have been compelled to abandon the practice of surgery.

Less than one hundred years ago there came a dramatic change. Surgical practice was transformed by the two greatest gifts given to the world by members of the Anglo-Saxon race, the discovery of anæsthetics and the discovery of antisepsis. It was a sad thing that these discoveries were made in the wrong order, for there was a ghastly interregnum of more than a score of years between the introduction of anæsthetics and the use of antiseptics, during which the surgeons were emboldened to extend the scope of surgery in directions formerly barred to them. The abdominal cavity, which had hitherto been opened only inadvertently, was deliberately explored with results which led Fergusson to exclaim that abdominal surgery should properly be described as abominable surgery, for the black pall of wound infection still overhung every hospital, virulent organisms were transferred from wound to wound by the hands of the doctors and nurses, and surgical mortality was appalling. Happily, Lister published his first papers on antiseptic surgery in 1867 and, with the spread of his doctrine throughout the world, surgical operations became comparatively safe. Nothing more aptly

expresses the value of his work, and, incidentally, nothing gave the Quaker soul of Lister greater pleasure, than the statement that by the discovery of antiseptic surgery more lives were saved in every year than Napoleon lost in all his wars; indeed, though mankind has since made great advances in the technique of wholesale slaughter, it is probable that the simile can be safely amended to conform to the standard set in the recent Great War.

It requires little imagination to picture the dramatic changes produced in the conditions of surgical practice by the use of the powerful weapons of anæsthesia and antisepsis, and the equally dramatic adjustment enforced in the field of surgical education. It became possible, providing that an early operation was performed, to save the lives of the vast majority of those afflicted by what are called surgical abdominal emergencies. To achieve this end, it became necessary to educate every member of the medical profession in the performance of operations of this type, because it was recognized that it was better for such a patient to be operated upon by the worst surgeon in the world during the first few hours of his illness than, after the lapse of two days, by the best. At the same time, as the scope of surgical work became rapidly broader and deeper, it was necessary to provide increased educational facilities for those who wished to practise surgery.

Those in charge of surgical education today must, therefore, concern themselves not only with the education of surgeons in the refinements of surgery, but also with the education of the general body of the profession in the rudiments of surgery. But this is not all, for, mainly in the last two decades, two other troublesome problems relating to surgical work have arisen. The first of these problems is the inevitable result of the rapid increase in the sum of surgical knowledge. There is no doubt that the day of the so-called general surgeon is ending and that we are witnessing the dawn of specialism in the various departments of general surgery. It is, therefore, necessary to determine the degree of general surgical education which should precede entry into a narrow specialty. The second problem is more difficult and arises from the fact that some men, who have had a surgical training sufficient only to justify them in performing urgent operations when the services of a skilled surgeon are unavailable, have been tempted beyond their strength partly by the glamour which, unhappily, is still attached to surgical work, and partly by the financial rewards which attend it, and have undertaken deliberately planned surgical operations of some difficulty on patients who could easily have been transferred at leisure to one more skilled in these procedures. The necessity for finding a solution to these various problem led to the foundation of the Royal Australasian College of Surgeons ten years ago, for it was believed that efficient direction of surgical education and control of conditions of surgical practice could be better accomplished, throughout Australia and New Zealand, by the formation of an Australasian body, directed by surgeons, than by requesting various existing academic and medical bodies in the separate Australian States and in the Dominion to undertake this work.

It was perhaps unfortunate that no name other than "College of Surgeons" could be found for this new body, for this term naturally implied that the functions of the Australasian College would be similar in character to those of the ancient colleges of Great Britain, which are in competition with the university, because they are examining bodies, bestowing qualifying diplomas similar to the university degree of bachelor of surgery and senior diplomas or fellowship equivalent to the mastership in surgery of a university. The Royal Australasian College of Surgeons has no desire to usurp similar functions, for, broadly speaking, its work begins where that of the universities ends. The possession of a fellowship diploma of the British type, or of a master of surgery degree, ensures that the recipient has attained a high standard of academic knowledge of his subject, but it does not necessarily ensure that he is a good practical surgeon, skilled in the actual conduct of operations. A candidate can present himself for examination for the master of surgery degree two years after graduation and the only practical training which is obligatory in the case of candidates for the English fellowship is six months' continuous service as a house surgeon in a general surgical ward of an approved hospital. It, therefore, occasionally happens that these qualifications are obtained by men who have no practical knowledge of operative work. It is the aim of the Australasian College to make sure that all those who are granted its fellowship shall have enough practical knowledge to justify them in operating upon human beings. Therefore, while recognizing that the acquisition of a senior surgical qualification must form an essential part of the education of a surgeon, it requires candidates for its fellowship to superimpose a period of apprenticeship training in practical surgery upon the more academic training required for these qualifications. Far from competing with universities, the Australasian College, in order to achieve its aims, must cooperate with them. In point of fact, as soon as the initial doubts concerning its objects, which had been aroused by misinterpretation of the meaning of the expression "College of Surgeons", had been allayed, there has been nothing but cordial cooperation between the College, the universities, and all other bodies concerned with the control of surgical education and the conditions of surgical practice in Australasia. It has been generally recognized that the dramatic alterations in the practice of surgery, to which I have already referred, have led inevitably to the creation of such a body as the Australasian College. Nor is this development peculiar to this country, for, in 1912, the American College of Surgeons was founded to cope with similar problems in the United States of America,

and, five years ago, a college was created in Canada. No modern college of surgeons can be content to function solely as an examining body. It must provide the community with an adequate supply of well-trained surgeons, it must foster research in surgery, it must supervise the conditions under which surgical work is undertaken, and it must inspire in all surgeons a spirit of conscientiousness in the performance of this work. All these activities should be included in the true meaning of the term "surgical education", and I propose to describe some of the work which the Royal Australasian College of Surgeons is doing in these various directions.

The first essential in providing a supply of well-trained surgeons is to define the course of training which these men must undergo. In the first ten years of its existence the Australasian College has had the unique experience of conducting a practical test of two separate and distinct systems of surgical training. When it was founded, it decided to adopt a system of admission similar to that of the American College of Surgeons, with the proviso that, after the lapse of a period of five years, all candidates for its fellowship should possess an academic senior surgical qualification. This system dispensed with any form of examination, but required each candidate to submit full data concerning his educational opportunities, his medical training and post-graduate work, and his literary efforts, and, further, to give the names of not less than five personal referees. Each application was then referred to the appropriate State Credentials Committee, which voted to accept, postpone or reject the applicant. The application, together with the decision of the Credentials Committee, was then considered by the Council of the College, which decided whether to reject or accept the candidate forthwith or to require him to submit a series of case histories of personally conducted operations.

Six years' experience of this system of admission convinced us that, in our hands, it was unworkable. The various Credentials Committees held widely differing views about the degree of surgical knowledge and ability which justified admission to fellowship; the confidential reports received from men, whose names were supplied by each candidate for the purpose of personal reference, were frequently diluted by benevolence and occasionally acidulated with malice; members of the Council had to sit in judgement upon men whom they had never seen and, finally, it was found almost impossible to determine the ability of a candidate by the perusal of a series of case histories submitted by him. There were, therefore, unmerited admissions and unjust rejections, and it was apparent that, unless this system was abandoned, the College would suffer greatly in prestige and thus would be unable to achieve its aim of supplying the community with well-trained surgeons.

Four years ago new regulations governing admission to fellowship of the College were adopted by the Council. These regulations are published in the handbook of the College, and it is therefore unnecessary for me to describe the requirements in detail, but some of the salient features merit emphasis. Each candidate must have had a minimum post-graduate training of five years' duration and must have obtained an academic qualification in the shape of a senior surgical degree or diploma which entails a primary examination in anatomy and physiology and a final examination in surgery. He must also have served a period of apprenticeship to a senior surgeon in the operating theatres and wards of an approved hospital, additional to the time spent therein as a house surgeon. The practice of attempting to qualify by performing a series of operations, unaided and unsupervised, is viewed with disapproval. Credit is given for work done in university departments, in research and in other countries. The course of training is not absolutely standardized and it is possible for each candidate to vary it in accordance with his desires and opportunities, but a period of apprenticeship is obligatory. When his training is completed, the candidate is examined by a Board of Censors of the College, but there is an important proviso that no candidate can appear before this Board until his course of training has been approved by the Censor-in-Chief of the College. Full details of the work done by each candidate are supplied to the members of the Board of Censors, together with reports from the surgeons who have supervised his training. The examination is *viva voce* in type and the questions are so framed that the answers cannot be given "out of books". Candidates who have been approved by the Board of Censors must apply to the Council for admission as Fellows and the Council reserves to itself the right of rejection on ethical grounds. There is no doubt that this curriculum is more searching and severe than that imposed by any other system of post-graduate surgical education, but, in this respect, the College is conforming to the tradition, founded by Halford, when, in 1863, he adopted a more severe undergraduate curriculum than any other in existence at that time. Four years' experience of our system of admission to fellowship has sufficed to convince us of its value. It is very encouraging that such an authority as Professor Edward Archibald, of Canada, in a presidential address to the American Surgical Association last year in which he reviewed the systems of surgical education in America, Britain, France, Germany and Australasia, should have stated that, in his opinion, the Australasian system was the best. It is also encouraging that the newly formed American Board of Internal Medicine should have adopted a similar system of training for specialists in this field. There is still a considerable misconception about the Australasian regulations, as is shown in a recent leading article in *The British Medical Journal*, in which the writer suggests that we have abandoned the British system of training and have steered a course which brings us nearer

to the American method. He goes on to explain that, in England, it is customary for aspirants for surgical appointments first to obtain a higher diploma and then to undergo a training in practical surgery. I hope that I have made it clear that the Australasian system closely resembles the British system with the important difference that what is customary in England has been made obligatory in this country.

A modern college of surgeons cannot afford to "rest on its oars" when it has devised a system of surgical training, for it is equally its duty to do everything possible to help aspiring surgeons to undergo this training. As the College has made the acquisition of a senior academic degree an essential part of its curriculum, its first task was to urge that there should be uniformity in the regulations governing admission to these degrees in the Australian universities. This was accomplished five years ago, the various universities agreeing to adopt regulations providing for a primary examination in anatomy and physiology. It should be noted that undergraduates are excluded from the primary examination for these degrees, as the university authorities thought it desirable to compel those men who wished to become surgeons to revise their knowledge of the basic sciences of anatomy and physiology after graduation. After this agreement had been reached, the College naturally defined a senior surgical qualification in its regulations as one which embraced these two examinations. At the same time, it safeguarded the interests of universities and colleges in which a senior degree or diploma could be obtained by one examination, by enacting that the two examinations need not necessarily be passed in the same university or college of surgeons. Another step in a similar direction was the request made by the College to the Council of the Royal College of Surgeons of England that it should hold its primary fellowship examination in this country. As you know, this request was granted and an examination was held here in 1931, both colleges agreeing to share the financial responsibility involved in the undertaking. The examination was repeated three years later and notice has recently been given that another examination will be held in Sydney, Melbourne and Dunedin in November, 1937. This scheme is not only of great imperial significance as a link between Australasia and the Mother Country, but also has the advantage that successful candidates must go to England to sit for the final examination and are thus brought under the influence of the great traditions of British surgery. On each occasion in which the examination was held here, 50% of the candidates were successful, a much higher percentage than is usual in this examination, a fact which, I am tempted boastfully to point out, is an indication of the high standard of medical education in this country. The opportunity of sitting for this examination in their own country is welcomed by Australian and New Zealand students, as is shown by the fact that in 1934 sixty-eight candidates presented themselves. The success of the scheme is a striking illustration of the value of cooperation between the English College, the Australasian College and the Australasian universities in the promotion of better surgical education.

I have already emphasized the importance of a period of surgical apprenticeship in the training of a surgeon and have explained that this forms an essential part of a curriculum for Fellowship of the Royal Australasian College. While the College is prepared to recognize suitable training of this character carried out in other countries, it was obviously necessary to provide adequate facilities in this country. In response to a request from the College, the great majority of the large clinical schools in Australia have created positions of this type. At the Royal Melbourne Hospital these men are known as "surgical associates" and one is appointed, providing that a suitable applicant is available, to each of the six surgical "firms" at the hospital. Each associate must possess a senior academic degree or diploma and must be personally acceptable to the surgeon with whom he is to work, an essential provision in an apprenticeship system. His duties are to assist at major operations, to help in the ward work and in the out-patients' department, to share in teaching duties, and to perform such operations as are delegated to him by the honorary surgeon. Surgical associates can hold office in any one "firm" for a maximum period of two years, and the positions are non-resident and unpaid, though it has been the custom to give these men a small gratuity from the clinical fund of the hospital as a reward for their teaching work.

Similar regulations are in force at the Alfred Hospital, Melbourne, which was the first hospital to make these positions available, and at Saint Vincent's Hospital. During the past few years, thirty men have undergone this training in Melbourne. Fears urged at the inception of the scheme that the presence of a surgical associate would curtail the privileges of the house surgeon have proved groundless. The success of this system of surgical apprenticeship must depend upon the members of the honorary surgical staff of each hospital, for these men must select and encourage the most promising of their juniors to qualify for this training. Those who fail to do so lay themselves open to the charge of suppressing their juniors lest these should deprive them of portion of their private work. It is to be hoped that these positions will soon be filled in all the large metropolitan hospitals and that it will, before long, be possible to extend the apprenticeship system to the large base hospitals in the country.

The next problem which confronted the College was that of the expense to candidates involved in this training. It was feared that the cost of undertaking a five-year period of post-graduate work, following upon that of a six-year period of undergraduate work, would deter many men from

qualifying as well trained surgeons. These fears are groundless to some extent, as is shown by the fact that, in the decade in which the College was founded, ten men in each year in Australasia voluntarily undertook a course of training of the type now made obligatory. A number of positions as senior resident officers in hospitals and as demonstrators in university departments are usually occupied by men who are undergoing special training of this and other types and who are prepared to live on the small income paid for this type of work. Though the great majority of our graduates are endowed with zeal, initiative and the spirit of self-help, it must be admitted that it will be necessary to supplement these opportunities by the provision of bursaries for suitable candidates for the Fellowship of the College, if an adequate supply of well-trained surgeons is to be maintained. It is significant and gratifying that the College has already received much help in this direction. The late Gordon Craig, a member of its first Council, has left it a munificent bequest of approximately £70,000, which, when it becomes available, will be used to foster surgical education and research. The family of the late Sir George Syme, its first President, have endowed a research scholarship, and T. F. Ryan, a Fellow of the College, has given it £5,000 to provide two surgical scholarships of £100 each, open to students graduating from Saint Vincent's Hospital and the Royal Melbourne Hospital, respectively. Finally, the College devotes part of its income for this purpose, thus adopting the appropriate principle that dues paid to it by the surgeons of today should be, in part, devoted to the education of the surgeons of tomorrow. It is to be hoped that many other surgeons will follow the example set by those to whom I have just referred and that the College will never be forced to adopt the habit, common in Australia, of appealing to the Government for assistance. It is necessary to emphasize the fact that the subsidies provided by the College will help research in universities and similar institutions, as the men who are given bursaries will work in these places in addition to the hospitals. I hope that in the future the Institute of Anatomy, in which we meet tonight, will benefit in this way. It is the present policy of the College to promote research in established institutions in preference to creating a research institute of its own.

Having described the activities of the College in formulating a curriculum of training in general surgical work and in assisting young men to undertake this training, I must now refer to the question of specialism in surgery. There have long been specialists in gynæcology, but, more recently, specialists have appeared in urinary surgery, in orthopædics, in brain surgery, and, within the last few years, in thoracic surgery, to mention only a few of the various branches from the main surgical trunk. The College believes that it is important that all who specialize in these directions should have a sound knowledge of general surgery as a foundation for the special knowledge required in each specialty, and, therefore, it does not bestow a fellowship for work in any of these departments, but requiries all candidates to undertake a training in general surgery. Pursuing this argument to its logical conclusion, it is obviously desirable that specialists in surgery should have some knowledge of general medicine, and it has been suggested that some years' work in general practice should form part of the education of a surgeon. The reason for this suggestion lies in the fact that some specialists tend to become narrow in their outlook and to attach disproportionate significance to symptoms involving the portion of the body in which they specialize. Though such men would undoubtedly benefit by experience gained in general practice, it is impossible to embody such a requirement in a post-graduate surgical curriculum. There are many difficulties in the way, of which two of the more important are the time required for such a training and the fact that, during this time, the young surgeon would be unable to obtain experience in surgical technique at an age when this is most valuable. I believe that the real solution of this problem strikes at the root of our system of medical education. Undergraduates at our clinical schools devote more attention to the study of medicine than to the practice of medicine. By this I mean that, though they gain a sound knowledge of the symptoms of organic disease and of the scientific methods of investigating the cause of these symptoms, they are not taught that each patient is not only a case, but is also a human being. It should be more forcibly impressed upon them by their teachers that, for example, the treatment of an organic surgical disease does not end with the performance of a successful operation, but that it is also necessary, in order to complete the cure, to pay attention to the psychological make-up and the economic environment of each individual patient. They should also be taught that symptoms identical with those of an organic disease can be caused by maladjustment to conditions of life, and that it is actually harmful to remove the appendix or the gall-bladder from a patient whose symptoms can be effectively alleviated only by providing a long holiday, a new husband, or a larger income. These lessons are learned soon enough in the hard school of practice; surely there is no reason why they should not be learned by undergraduates. It would help in this direction if a small book, "The Care of the Patient", by Francis Weld Peabody, late Professor of Medicine at Harvard, was made a textbook for students in every clinical school, for the book is an elaboration of the philosophy expressed in its last sentence: "One of the essential qualities of the clinician is interest in humanity, for the secret of the care of the patient is in caring for the patient."

Education of specialists in general medicine and surgery would be less important if every patient realized that it is better to consult his family doctor

before consulting a specialist. The advice of the family doctor, the general practitioner, is of great value to the patient in the selection of a specialist, and of equal value to the specialist in the treatment of the patient. It is most unfortunate that the proper relationship between specialists and general practitioners has been upset to some extent by specialists who engage in general practice, and by general practitioners who try to be specialists. It would add greatly to the prestige of medicine if every practitioner would remember that "the cobbler should stick to his last".

The relationship between specialists in surgery and general practitioners is complicated by the fact that, as I have already shown, it is necessary to provide some education for all members of the medical profession in the performance of urgent surgical operations. Every medical student, as soon as he has graduated, is legally entitled to perform any surgical operation, despite the fact that he is not competent to do anything more difficult than suture a cut or open a boil. It is therefore essential that all recent graduates should gain practical experience by acting as resident medical officers in large hospitals and that they should be given opportunities to acquire some proficiency by performing operations, under supervision, during their period of office. At the Royal Melbourne Hospital it is the practice to delegate operations of the appendix-hernia type to the house surgeons after they have served in this capacity for one month. All operations must be performed in the presence of an honorary surgeon of the hospital. In each year approximately three hundred operations of this type are performed by house surgeons, who thus gain some confidence in the operative treatment of surgical emergencies. At the same time, it is impressed upon these men that this training does not justify them in embarking on a surgical career, but that, if they wish to do so, they must emulate the example of the surgical associate and undergo a long apprenticeship. The Australasian College proposes to share in this work by affording opportunities for post-graduate education in surgery to all members of the medical profession at the post-graduate school which is being established at Prince Henry's Hospital, Melbourne. It will also be possible for men, who have been in general practice and wish to qualify as surgeons, to undertake the full course of training in this institution.

The necessity of educating every doctor in the elements of surgery so that he can cope with surgical emergencies in the absence of a skilled surgeon obviates any possibility of restricting surgical work at present to those who have had a special training, but signs are not wanting that a change is imminent and inevitable. In the first place, modern surgical work involves the use of extensive equipment which can only be provided in large hospitals. This equipment, which becomes more costly and more complex year by year, is essential for success in many operations and is invaluable in every operation. This development is not peculiar to surgery; it permeates almost every aspect of modern civilization, and I can best exemplify what I mean by suggesting that the surgeon of the future, who is deprived of the use of this equipment, will be almost as impotent in the struggle against disease as were the Abyssinians when opposed to the modern methods of warfare of their recent adversary. It therefore seems obvious that a time is rapidly approaching when surgical work must perforce be restricted to hospitals which provide this complicated equipment. In the second place, rapid improvements in transport facilities, particularly by air, are revolutionizing the conditions of practice, so that it will soon be possible to remove all patients in need of urgent surgical attention, wherever they may be, to a properly equipped hospital in an incredibly short space of time. Those of you who have read a book called "Can the Doctor Come?" by Walquist, will appreciate how this is done in Lapland today and, of course, the achievements of the Aerial Medical Service in the inland areas of this country, in which we take such justifiable pride, are an even more forceful indication to us of future developments. Deaths due to modern transport occupy an imposing place in mortality statistics today, so that it is appropriate that the life-saving potentialities of this product of modern civilization should be developed. Finally, team-work or cooperation between surgeon, physician, bio-chemist, radiologist and pathologist is becoming increasingly important in all departments of surgery, including that of surgical emergencies. This team-work is possible only in large hospitals where staffs of specialists are available. I therefore believe that before many years surgical work, which, following Lister's discoveries, became the responsibility in greater or lesser degree of almost every member of the medical profession, will once more be restricted to those who have been specially trained to perform it.

Those in charge of surgical education should do everything possible to hasten the advent of these changes in the conditions of surgical practice, which will necessarily abolish bad surgical work in ill-equipped hospitals. To achieve this end they must provide a supply of well-trained surgeons to fill positions at all large hospitals, they must willingly cooperate with all other bodies concerned with the administration of these hospitals, and with medical education in general, and they must preach the gospel that these changes are necessary and inevitable.

It is futile to preach the gospel to them who have not ears to hear, and, for this reason, I do not think that education of the general public in this matter would serve a useful purpose. In the medical sense, many even of the intelligent section of the public are living in the days of mysticism and witchcraft, and few of them are able to appreciate the importance of the changes to which I have referred.

To most of them a hospital is a hospital and a doctor is a doctor. They fail to realize that there is as much difference between a well-equipped and a badly-equipped hospital as there is between the motor car of 1901 and that of today. Indeed, I have been forcibly impressed by the fact that the majority of patients ask nothing more from a hospital than that it should be conveniently situated for their visitors. They cherish the extraordinary belief that a doctor who does not do surgery is only half a doctor, an attitude of mind which forces many men to undertake surgical work. The blame for much of this ignorance may justly be laid upon the shoulders of the medical profession, which has made little or no attempt to instruct the public in medical matters; but the fact remains that this would be at the best a very slow process. It is quicker, simpler and more effective to reform conditions of practice by educating all members of the medical profession in the need for these reforms—a task made easier by the fact that the vast majority of my colleagues hold fast to the tradition that the welfare of the public is the first aim of the profession.

It will undoubtedly encourage men to undergo an adequate surgical training if it is apparent to them that increased opportunities are to be afforded to well-trained surgeons. This, as I have said, can be achieved only be cooperation between the various bodies controlling all aspects of this work, so that united efforts to promote better conditions can be made. A most important step in this direction has recently been taken by the Charities Board of Victoria, which has adopted a plan for the staffing of base hospitals, suggested by its Chairman, Mr. J. Newman Morris, a Fellow of the Australasian College. This plan has been drafted with wisdom and with vision, and provides for the classification of the staffs of these hospitals into surgeons, physicians and other specialists. It gives preference, in the making of these appointments, to men holding special qualifications in each specialty, and makes the possession of such qualifications obligatory in five years' time. It establishes an Electoral Board, composed of representatives of the University of Melbourne, the Royal Australasian College of Surgeons, the Association of Physicians, the British Medical Association, the Charities Board and the Hospital Board concerned, which shall have power to make these appointments. It also provides for the hospitals subsided services of pathologists and radiologists. It is obvious that this scheme supplies a great incentive to men to undergo special training and thus to obtain qualifications which will be essential for appointment to the base hospitals in large country centres. It is equally apparent that the scheme conforms to the general trend of development of surgical work in the future to which I have already referred.

It has been argued that this development of specialist practice throughout the country will imperil the interests of the family doctor and that general practice, rightly termed "the backbone of the profession", will assume a less important rôle. Sir Farquhar Buzzard, in his presidential address to the British Medical Association in Oxford this year, in referring to this and other changes in the conditions of practice, states that he fails to see why general practice should not assume a more important rôle in the future than it has done in the past.

> The saving of life has become the job of institutional team work; the saving of health and the prevention of illness, in the long run of far greater interest to the welfare of the community, must be initiated by the general practitioner in the home.

He also emphasizes the importance of linking up the general practitioner with hospital work, so that:

> His personal interest in the welfare of his flock will not be dissipated by losing touch with its members.

There is no doubt that, in any reform of conditions of practice, the interests of the general practitioner must be conserved, but there is also no doubt that this does not necessarily entail the delegation of surgical work to those general practitioners who have not had special training for it.

Finally, may I point out that no system of surgical training can be successful if the pupils are not inspired by their teachers to be conscientious in the pursuit of knowledge, careful of the welfare of those whose bodies are entrusted to them, and courageous in confronting the dangers and difficulties encountered in their fight against disease. What better words could I use to emphasize the need for, and the reward of, these qualities than those used by George Britton Halford in an address to his students seventy-three years ago:

> You are called to a fellow creature in the agonies of death; he points instinctively to his throat, for he scarcely sees you; his last gasp is at hand and now he is insensible. You hesitate, and he is dead; but you operate and he lives; and you return home thanking God for the power He has given you to save.

THE MECHANISM OF VISCERAL PAIN.[1]

By HERBERT J. WILKINSON,
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MANY years ago, while still a student, I was examining a patient who complained of a pain in the epigastrium and noticed that when I placed my hand on this region there was a spot which felt definitely cooler to the touch than the surrounding areas. On careful palpation I was able accurately to map out this spot with the dermograph pencil, and was interested to discover that the patient, on being tested many times, always located the pain in this spot. On many occasions after this, in cases of pain which was not due to local inflammation, I made a practice of trying to discover the painful area without interrogating the patient by simply

[1] Read at a meeting of the Royal Australasian College of Surgeons, Brisbane, on August 14, 1936.

detecting the area of skin which appeared to me to be cooler to the touch. I would then ask the patient if he felt the pain there, and he often seemed mystified and wondered how it was that I could detect the position of the pain.

Soon after this, Professor A. E. Mills, who was the professor of medicine in Sydney at the time, informed us in one of his lectures that he believed that anoxæmia or oxygen want was an adequate stimulus for pain, and later embodied his ideas in a published paper.[1]

These two facts, namely, decreased temperature in painful areas and anoxæmia as the adequate stimulus for the production of sensations of pain, were at once connected in my mind, for it seemed to me that as the decreased temperature was probably due to vaso-constriction, this would no doubt also cause anoxæmia, and thus the patient would experience pain.

In studying Mackenzie's theory of the mechanism of visceral pain, which was the orthodox teaching, it appeared to me that my observations provided another explanation of the mechanism.

Since graduation, therefore, when giving my students an account of Mackenzie's theory, I have given them another theory based on my own observations; and this was not challenged until I put it to my post-graduate classes early last year.[2] Some of the surgeons who attended my classes were acquainted with the book of John Morley on "Abdominal Pain" and seemed inclined towards his view. This led to further experimental investigation and a review of the literature on the subject, with the result that I believe my views were more firmly established.

Before stating my theory in full, however, it would be well briefly to review the work of past investigators and to see what facts have emerged as a result of their investigations.

The first recorded significant observations which threw any light on the problem of visceral pain were published in 1763 by Haller.[3] He found that ordinary stimuli applied to the viscera did not give rise to sensations of pain and concluded that there was no evidence that the viscera were sensitive to pain.

No attempt seems to have been made at the time to put forward any explanation of how disorders of apparently insensitive viscera could cause pain, and these observations of Haller appear to have been forgotten until rediscovered by surgeons after the introduction of the operation of colostomy of the pelvic colon and the introduction of operative procedure under local anæsthesia. The surgeons soon discovered that the viscera could be touched, cauterized and incised without the patient's experiencing any pain whatever. The problem at once presented itself: "How could visceral organs which were apparently insensitive to ordinary stimulation give rise to pain?"

It is true that so long ago as the early sixties of last century John Hilton,[4] in his lectures to the Royal College of Surgeons from 1860 to 1862, made some interesting statements which show that he had given thought to the question of the mechanism of visceral pain and the importance of an exact knowledge of the distribution of the nerves connected with any one segment of the brain stem or cord. It is by following the injunction of Hilton, pronounced in the early sixties, that successive investigators have established a mass of facts out of which now emerges what I believe to be the true explanation of visceral pain.

Although Hilton did not attempt to give an exact explanation, he nevertheless was the first to recognize the true course along which fruitful investigations should proceed.

In 1886, Ross[5] read before the Manchester Medical Society his paper on "The Segmental Distribution of Sensory Disorders", in which he stated that there were two kinds of pain due to visceral disorders, namely, splanchnic pain, which was felt in the organs giving rise to afferent stimuli, and associated somatic pain, felt in that part of the body wall connected with the same segment of the cord as the affected viscus. He said that this was due to the production of an "irritable focus" in the cord, produced by strong afferent splanchnic nerve stimulation.

Ross was the first, then, to put forward the exact idea that visceral pain was sometimes "referred" pain.

The work of Ross profoundly influenced Mackenzie,[6] who, in spite of a very busy practice in Lancashire, lost no opportunity of investigating the problem of visceral pain and who accumulated a mass of facts on which he subsequently based his theories; these, even up till today, are widely accepted and are still presented in our textbooks of medicine and surgery.

Mackenzie quickly recognized the significance of Ross's somatic or "referred" pain, but was doubtful of his splanchnic pain, which he considered was also really "referred". He found, for example, that the pain of gastric ulcer, when limited to the epigastrium, and which Ross considered was splanchnic pain, remained fixed in deep inspiration and therefore could not be considered a true splanchnic pain. This led to his observations on areas of cutaneous hyperalgesia, followed soon by those on what he called "protective rigidity". He described the symptoms of pain and "protective rigidity" which occur in consequence of disease of the viscera as due to a viscero-sensory reflex and a viscero-motor reflex respectively.

According to Mackenzie, the mechanism of visceral pain or the viscero-sensory reflex is as follows [see Figure I (*a*)]: Impulses arising in a viscus, *X*, are conveyed by sympathetic afferent nerves to the central nervous system, X^1, when ordinarily no sensations are felt. If the impulses are of such a nature that on reaching the central organ they spread to other cells, including the cells in the somatic pain pathway, then they reach the sensorium, *S*, and give rise to pain which is referred to *B*, the area of the peripheral distribution of the

pain nerves. Similarly, the viscero-motor reflex [see Figure I (*b*)] is due to the visceral afferent impulses spreading to the anterior cornual cells giving rise to increase in tone in striated somatic muscle, *C*, of the abdominal wall. Mackenzie observed that this increased tone varied in intensity, and, when it was scarcely perceptible, it could be increased by rubbing gently along the muscle.

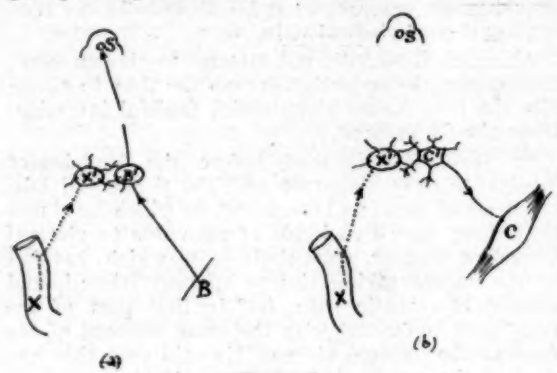

FIGURE I.

Diagram after Mackenzie, to illustrate his (a) viscero-sensory reflex, (b) viscero-motor reflex.

Meanwhile, Lennander[7] in 1901 published the results of a series of experiments on the sensitiveness of the viscera to ordinary manipulation and showed that although the viscera themselves were insensitive, the parietal peritoneum and subserous connective tissue were very sensitive. As a result of these investigations he formulated the theory that painful sensations from disease of the viscera originated in the parietal peritoneum and its subserous layer, both of which, around the whole of the wall of the peritoneal cavity, are richly supplied with cerebro-spinal (somatic) nerves. Pain of inflammatory lesions was, he said, therefore due partly to contact of inflamed parts with sensitive parietal peritoneum and partly to irritation of sensory cerebro-spinal or somatic nerves in the root of the mesentery by lymphadenitis and lymphangitis. He further considered that pain of intestinal colic was partly due to stretching of the parietal attachments of the bowel at the root of the mesentery, and partly to pressure on the parietal peritoneum of the anterior abdominal wall by contracting coils of bowel as they erect themselves in tetanic spasm.

As Morley says:

> In denying the existence of any afferent splanchnic nerves capable of transmitting painful stimuli, he ignored important histological, experimental and clinical evidence that such nerves existed. Thus, though his observations of fact are of prime importance, his theory which he based upon them has failed to explain many other facts concerning abdominal pain that have come to light since his original researches.

Mackenzie agreed with Lennander that pain did not arise in the viscera, but, unlike Lennander, believed that the viscera were supplied with afferent nerves. Also, like Lennander, he recognized that pain may be due to invasion of the body wall, although he expressed himself as unable to differentiate in practice between this and the viscero-sensory reflex. He says (page 204):

> In many visceral ailments the inflammatory affection extends and involves the sensitive structures of the abdominal wall, and with this extension a new train of symptoms arises which may lead to wrong inferences if these differences in the sensibility of the structures are not clearly kept in view. One can easily see how appendicitis may give rise to symptoms which are entirely confined to the reflex group, until the inflammation extends to the abdominal wall, when another series of symptoms may arise which are produced by a different mechanism, and are detected in the immediate region of the inflammation.

Further contribution to this subject was made by Head[8] in 1893. Head fully accepted Ross's theory of "referred" pain and made it the basis on which he constructed his detailed account of the splanchnic innervation of the viscera. Later in 1905[9] Head's paper on "Protopathic and Epicritic Sensation" appeared, in which he brought forward the idea that the viscera as well as the skin are supplied with nerves of protopathic sensation. Nevertheless he supported Ross's and Mackenzie's theories of "referred" pain and the viscero-sensory reflex respectively, and was no doubt influenced in this respect, as Morley says, by his study of the phenomenon of allocheiria in cases of *tabes dorsalis*, in which a stimulus applied to a skin area of low impaired sensitivity is referred to an area of normal sensitivity. It appeared to Head that in a similar way stimulation of the relatively insensitive viscera may lead to pain referred to the more highly sensitive superficial structure of the abdominal wall.

A further step was reached in 1911, when Hurst[10] proved that although the gastro-intestinal tract was insensitive to cutting, pinching or burning, it was sensitive to an adequate stimulus. This was a great advance. Hurst showed that, provided the adequate stimulus was applied to the viscera, pain could be elicited. He found that the adequate stimulus was increased tension on the muscular wall, due to either stretching, dilatation or an excessive contraction. Hurst believed, therefore, that there were: (i) true visceral pain due to increased tension in the viscera and vaguely localized to the centre of the abdomen and not referred to sensory areas in the abdominal wall, and (ii) "referred" pain as taught by Mackenzie.

In 1920, Hunter,[11] of Sydney, in his essay for the Sandes Surgical Prize for final degree candidates, elaborated Mackenzie's theory and gave a more detailed account of the neurological mechanism probably involved and offered further support of Mackenzie's theory.

In 1926, Lemaire[12] made the interesting discovery that, when he induced local anæsthesia of the whole thickness of the abdominal wall by injecting 0·5% "Novocain" in four patients with abdominal pain, a prompt disappearance of the pain, tenderness and muscular rigidity occurred in all four cases. Subsequently he showed that it was only necessary to give a subcutaneous injection of

the "Novocain". These experiments led Lemaire to support the theory of the viscero-sensory and viscero-motor reflexes, but he said that the impulses passed into the pain pathway, not in the cord (Mackenzie), but in the posterior root ganglia.

In 1928, Weiss and Davis[13] published the results of similar experiments and submitted what they believed to be direct experimental support of Mackenzie's theory of "referred" pain. Their argument was that if the conception of "referred" pain was correct and visceral pain was caused by the normal afferent impulses from the skin entering a cord segment which had become abnormally irritable as a result of visceral bombardment, it would follow that the cutting off of these painfully felt afferent impulses from the skin should result in the abolition of pain. They were able to show that in patients suffering from distinctly localized severe visceral pain, infiltration with "Novocain" of the corresponding painful skin areas caused prompt relief of pain. They further showed that in normal subjects, when localized pain was induced experimentally by distending certain portions of the œsophagus, infiltration of the skin over the localized painful area caused relief, but the pain appeared in another skin area. When the effect of the anæsthesia wore off, the pain returned to the original painful area. This "migration of pain" on anæsthetizing the painful area was also noticed in two patients with diseased viscera. This is an interesting observation and will be referred to again later.

They further discovered that occasionally a dull, unpleasant sensation, described by such terms as soreness, discomfort and pressure, but never as pain, was noticed after skin infiltration. They suggested that this might be evidence of protopathic sensibility of the viscera, as suggested by Head, but they could not be sure that it was not due to afferent impulses from outside the peritoneal or pleural cavity.

In 1931, Morley[14] published his valuable book on "Abdominal Pain", in which he states that he is:

> firmly convinced that true visceral pain exists and that, as Hurst has pointed out, it is usually the result of abnormal tension on the splanchnic afferent nerve endings in the muscular walls of the hollow viscera. It is in no sense referred to the superficial structures of the abdominal wall, and is a deep-seated central pain not accurately localized.

He writes further:

> When pure visceral pain occurs, as in the early hours of an attack of acute obstructive appendicitis, it is entirely unassociated with any tenderness, superficial or deep, or with any reflex muscular rigidity of the abdominal wall.

I have quoted Morley here in full, as I want you to have no doubt whatever about his contention. He says again that he:

> believes that the phenomena of deep and superficial tenderness and rigidity of the abdominal wall, so commonly observed in association with inflammatory disorders in the abdomen, are in no way concerned with the afferent autonomic system, but are entirely referred from the highly sensitive cerebro-spinal nerves under the parietal peritoneum.

This is his theory of peritoneo-cutaneous radiation and the peritoneo-muscular reflex (see Figure II). He denies entirely the existence of Mackenzie's viscero-sensory and viscero-motor reflexes. In stating his theory, he leaves open the question as to where the afferent impulses from under the peritoneum pass into the pain pathway.

In support of his theory that the afferent impulses arise in the sensitive layer under the parietal peritoneum, he conducted experiments to show how, in the case of gastric ulcer, the point of deep tenderness shifts with alteration of posture.

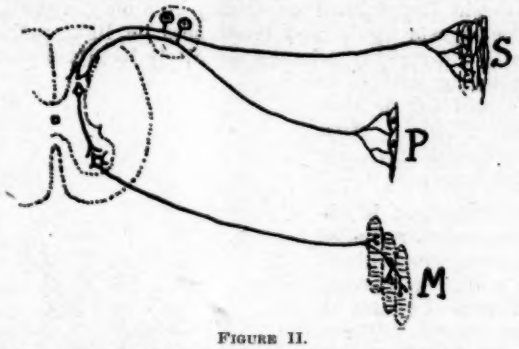

FIGURE II.

Diagram after Morley to illustrate his theory of peritoneo-cutaneous radiation and the peritoneo-muscular reflex. *S* = skin, *P* = parietal, *M* = muscle of the abdominal wall.

It is to be remembered that Mackenzie had contended that this area remained fixed, and since Morley's book appeared, Bolton,[15] in answer to Morley's claims, has given an account of observations which were made with the object, as he says, of affording evidence that pain and tenderness in patients with gastric and duodenal ulcer is of the visceral referred type as described by Mackenzie, quite apart from the question as to whether splanchnic pain occurs in addition. He gives evidence to prove that the points of tenderness did not always correspond to the position of the ulcer and definitely remained fixed in position, no matter whether the patient was erect or supine. The different results obtained by Morley and Bolton in these experiments are referred to again below.

So that we might get a clearer conception of the subject, it would be well to summarize the views expressed up to the present by the most familiar of the authors, as follows:

(1) Mackenzie: (*a*) denies the existence of true visceral or splanchnic pain. (*b*) says that visceral pain is usually due to the stimulation of visceral afferents and is "referred" to some superficial area (viscero-sensory reflex). This is referred to in this paper as the visceral referred pain. (*c*) does not deny the existence of pain arising from stimulation of the somatic nerves in the subserous tissue of the parietes, but did not elaborate on this idea.

(2) Morley: (*a*) defines true splanchnic pain as deep-seated, vaguely localized, mid-line pain, and that adequate stimulation of the visceral afferents always leads to pain of this nature. (*b*) says that other pain associated with inflammatory disorders of the viscera, and called by him somatic pain to distinguish it from true visceral pain, is

due to stimulation of the somatic afferents in the parietes (peritoneo-cutaneous radiation). This is referred to in this paper as the peritoneal referred pain.

(3) Bolton: does not deny the existence of true visceral or splanchnic pain, but says that pain and tenderness in patients with gastric or duodenal ulcer is of the referred type, thus supporting Mackenzie.

There are thus three conceptions, namely: (i) true visceral or splanchnic pain, (ii) visceral referred pain (afferent stimuli arise in the viscera), (iii) peritoneal referred pain (afferent stimuli arise in the parietes).

It is to be noted that true visceral pain as defined by Morley (see above) is unaccompanied by hyperæsthesia (heightened sensitivity to touch stimuli), hyperalgesia (pain and tenderness on light palpation) and rigidity, whereas definitely localized pain, occurring either in the mid-line or elsewhere, is often accompanied by these phenomena.

Further, it is obvious from recorded observations that pain due to visceral disorder is caused in two ways. First, there is the pain primarily due to adequate stimulation of the visceral or autonomic afferents, and brought about usually by states of increased tension in the viscus, however produced. Secondly, there is pain primarily due to adequate stimulation of the somatic afferents which arise in the parietes of the body cavities. This latter pain occurs when the parietal tissue is stimulated by an inflamed viscus which comes in contact with it.

I will now state my alternative theory of the mechanism of visceral pain, based originally on the observations made on associated alteration of the skin temperature.

The facts are briefly:

1. In cases of pain due to disorders of the viscera there is noticed a distinct lowering of the temperature in the skin over the seat of the pain. This was verified by skin thermometers.

2. The pain is relieved by infiltration of the "cool" area with 2% "Novocain".

It is emphasized that these observations were made on cases which exhibited not only the true splanchnic pain as defined by Morley, but the definitely localized pain often accompanied by hyperalgesia and protective rigidity.

From a consideration of these facts it seems to me that one is justified in concluding that pain due to disorders of the viscera is, in the observed cases, neither felt in the viscera nor is it referred to the body wall, but actually arises in the body wall. If this is so, then we must look for a different mechanism for its production than those already put forward.

The Theory of Viscero-Vasomotor Reflexes.

First let us consider the mechanism of the pain due to adequate stimulation of the visceral afferents (see Figure III).

Normal stimulation of the visceral afferents from the gut produces impulses which enter the cord via the posterior root ganglia and pass over to the neurones situated in the lateral cornu of the cord. Impulses are then discharged along the preganglionic sympathetic fibres to the ganglia of the prevertebral plexuses and thence along post-ganglionic fibres to the gut, causing inhibition and relaxation of the muscular walls. Normal stimulation of the visceral afferents merely causes reflex changes in the gut, and there are no well-defined alterations of consciousness and there are certainly no sensations of pain.

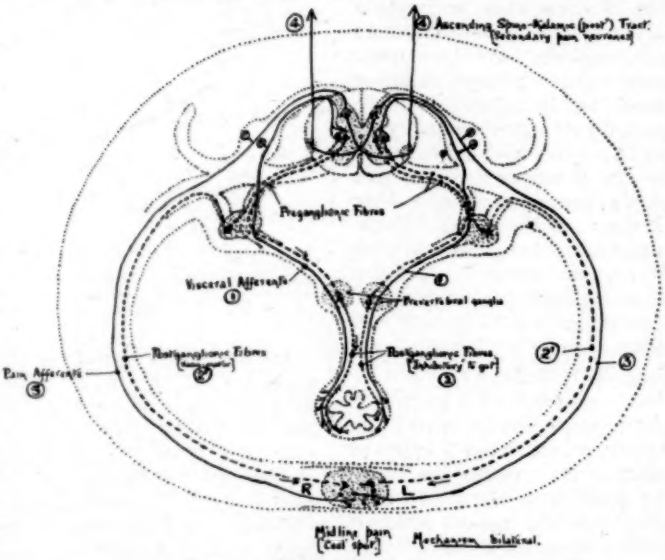

FIGURE III.

Diagram to illustrate the mechanism of the vaguely localized anterior mid-line pain associated with stimulation of visceral afferents put forward in this paper. Mechanism bilateral. See text. Normal reflex for inhibition of gut = $1 \rightarrow 2$; viscero-vasomotor reflex = $1 \rightarrow 2^1$; viscero-vasomotor reflex resulting in pain = $1 \rightarrow 2^1 \rightarrow 3 \rightarrow 4$.

More violent stimulation of the gut leads to increased bombardment of the sympathetic nuclei in the lateral cornu of the cord, with the result that the impulses pass over not only into those neurones ultimately connected with the gut, but into adjacent vasomotor neurones which innervate the peripheral vessels. This causes vaso-constriction and associated conditions in these areas, thus providing the adequate stimuli for the pain nerve-endings. Pain impulses thus arise in these superficial somatic areas and pass via the posterior root ganglia into the cord and thence via the secondary neurones of the pain path, the fibres of which run in the posterior spino-thalamic tract to the thalamus and thence on to the cortex. When this occurs we experience sensations of pain.

Briefly, the visceral afferents, when adequately stimulated, cause reflex vaso-constriction in the somatic peripheral structures and conditions thus arise here which lead to stimulation of the pain endings and to the production of pain sensations.

The pain is thus felt in the somatic tissue, not because it is merely "referred" here, but because the pain nerves are actually stimulated here. The lowering of the skin temperature is merely a manifestation of the vaso-constriction and associated phenomena which provide the adequate stimulation for pain impulses, and is not to be regarded as the cause of the pain. It is quite obvious that the mere lowering of the temperature by 1° or 2° C. is not the adequate stimulus for pain.

It might now be asked why the pain occurs merely in the mid-line instead of in the complete area of distribution of the vasomotor nerves of the segment or segments involved. A glance at the figure shows that the reflexes occur on both sides of the body, and, owing to the anastomoses of the vessels, it is in the mid-line that the phenomena will be most intense. Sometimes the pain is not exactly in the mid-line and may occur a little to the right or to the left of the mid-line. It is not unreasonable to assume that in different individuals the area of maximum conditions for the production of pain varies about the mean position, which is approximately in the mid-line. This question of the localization of pain, however, is more fully considered below.

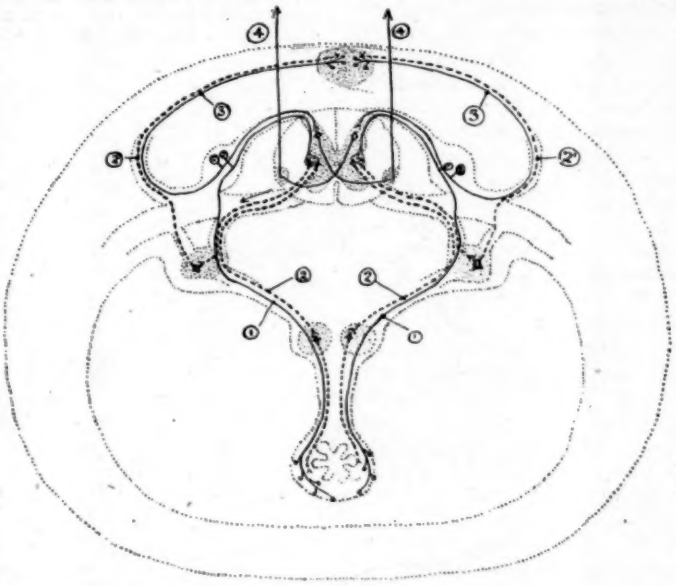

FIGURE IV.

Reflexes concerned in the production of pain in a posterior area. Normal reflex for inhibition of gut = $1 \rightarrow 2$; viscero-vasomotor reflex = $1 \rightarrow 2^1$; viscero-vasomotor reflex resulting in pain = $1 \rightarrow 2^1 \rightarrow 3 \rightarrow 4$.

This mechanism also explains why a pain is sometimes felt in the back (see Figure IV); in fact, the pain in the back is, in some cases, the only pain felt. Further, the combined sensations of pain in the anterior abdominal wall and in the back may give rise to the judgement of the pain as being deep-seated.

Another significant fact is that when the pain is felt only in the mid-line and a "cool" spot is identified and infiltrated with "Novocain", the pain appears to "migrate" to another part of the segment involved, as described by Weiss and Davis (see above) and which was noticed in my own experiments. This is due, I think, to the fact that the patient experiences only the more intense pain, and when this is abolished, the less intense pain, if it exists, is felt in another part.

Hyperalgesia and Protective Rigidity.

Morley has pointed out that true splanchnic pain is unaccompanied by hyperalgesia and protective rigidity, and postulates for the production of these a different mechanism. Since these conditions have also been observed by me to be accompanied by a lowering of skin temperature and can be abolished by infiltration with "Novocain", it seems apparent that they can be explained in the same way. When the conditions in which the so-called mid-line splanchnic pain occurs are sufficiently intense or have been in existence for some time, the skin becomes hypersensitive to touch stimuli (hyperæsthesia) and there is heightened sensitivity of the afferent nerves arising in the underlying connective and muscle tissue. On light palpation, therefore, we elicit the signs of hyperalgesia, and reflexes arising in the muscle tissue give rise to increased tonus in the muscles affected (protective rigidity). Mackenzie has pointed out that if the rigidity is only very slight, it can be elicited by gently stroking the muscle. The stroking obviously augments the reflexes for the production of this increased tonus.

Morley says that rigidity can arise only as the result of stimulation of the parietal somatic afferents (peritoneo-muscular radiation) and never occurs as a result of stimulation of the visceral afferents, as Mackenzie states (viscero-motor reflex); but Mackenzie has made it quite clear that rigidity does occur in visceral disorders in which the parietes cannot be involved. It appears to me that just as vasomotor phenomena lead to pain in the superficial tissues, they can also lead to pain and tenderness and reflex spasm in the underlying muscle tissue. This brings "protective rigidity" into line with so-called cramp in muscles consequent upon deficient circulation.

Pain and Rigidity due to Stimulation of Parietal Somatic Afferents.

We shall now consider the pain due to involvement of the parietal peritoneum in inflammatory conditions of a viscus, and brought about by stimulation of the somatic afferents. Examples of this type of pain are the secondary pain in appendicitis and shoulder-tip pain. Since here also there is alteration of skin temperature and the pain can be abolished by infiltration of the painful areas with "Novocain", it seems to me that the mechanism is similar to that described above. (See account of cases below.)

The only difference in the mechanism is that the somatic afferents from the parietes convey the primary impulses instead of the visceral afferents (see Figure V). Subperitoneal somatic afferents, on stimulation, reflexly cause vasomotor phenomena in the overlying skin and muscle tissue, setting up conditions for the production of pain stimuli and reflex rigidity, as described above. It is significant that even subcutaneous infiltration with "Novocain" abolishes the rigidity (Lemaire, Weiss and Davis). These authors, however, put a different interpretation on the result of their experiments than that which accompanying alteration in skin temperature leads me to put forward.

"Irritable Focus" in the Cord.

Weiss and Davis, in interpreting the results of their infiltration experiments, accepted the idea of Ross and Mackenzie of an "irritable focus" in the cord, which they believed was the result of abnormal visceral bombardment and which formed part of their mechanism for the production of "referred pain". Although my observations lead me to different conclusions with regard to the mechanism of visceral pain, I do not think we can completely dismiss this idea of an "irritable focus".

A careful examination of the connexions of the sympathetic system reveals that recurrent post-ganglionic fibres enter the cord via the roots of the spinal nerves, and it is quite possible that these are stimulated at the same time as those which pass to the periphery, and produce effects in the cord itself which tend to lower the resistance at the synapses in the part of the cord concerned. In working out the complete mechanism, therefore, for pain associated with visceral disorders, I do not think we should overlook the possibility of effects in the central organ itself. The complete picture may thus include heightened sensitivity in somatic structures *plus* the lowering of the resistance at the synapses in the cord.

With this presentation of the mechanism of pain associated with disorders of the viscera as due to associated reflex conditions set up in the superficial somatic tissues owing to excitation of the vasomotor neurones, let me describe observations and experiments made in typical cases.

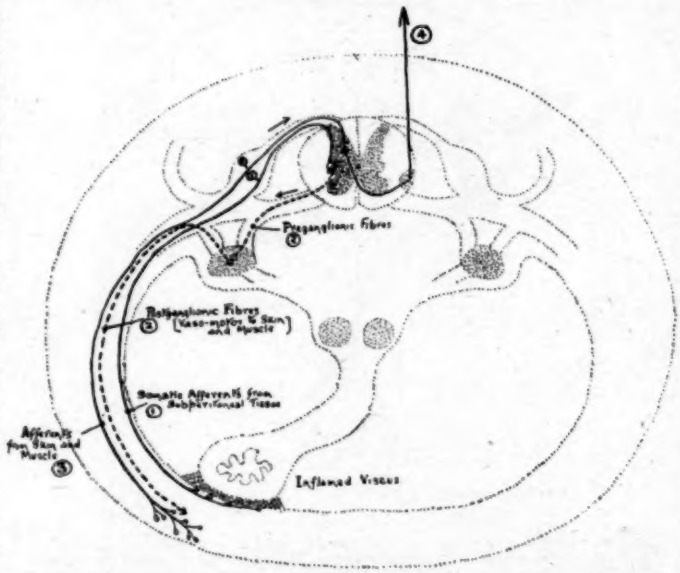

FIGURE V.

Diagram to illustrate the mechanism of definitely localized pain associated with the stimulation of peritoneal somatic afferents put forward in this paper. Mechanism unilateral. See text.

Patient A (diagnosis, appendicitis) complained of a deep-seated dull pain in the region of the umbilicus. There was no hyperæsthesia and hyperalgesia in this area. On careful palpation with a warm hand there was discovered just below the umbilicus an area of about 35·0 millimetres (one and a half inches) in diameter, which felt distinctly cooler than the surrounding skin[1] (see Figure VI). A skin thermometer (number 1) was placed so that the flat bulb rested in the centre of this area. This was covered with a thin layer of cotton wool and a small strip of "ZO" strapping was placed over all to hold them in position. Care was taken not to exert any pressure on the bulbs. Thermometers numbers 2, 3 and 4 were then similarly fixed on surrounding skin areas. The patient was covered with a blanket and after five minutes readings were taken. Thermometer number 1 showed a reading of 35° C., while the other thermometers showed readings of 36° C. This area was then infiltrated subcutaneously with a 2% solution of "Novocain" and the pain eventually disappeared. The patient, however, said that he could now feel a pain (*B*) in the iliac region. This second pain reached its maximum within from forty-five minutes to one hour after the injection of the "Novocain" into area *A*. On examination the patient exhibited a slight degree of hyperæsthesia and hyperalgesia in this region.

The thermometer number 1 over area *A* then gave a reading of 37·5° C., while the others remained constant at 36° C.

Within about one and a half hours after the injection the central pain began to appear again and gradually increased until, within two hours, it was the only pain felt. Thermometer number 1 over area *A* was now giving a reading of 35° C., and the others a reading of 36° C.

A reading was now taken over area *B* and was found to be 35·5° C. Area *A* was infiltrated again, and after thirty minutes, when the pain in area *B* was distinctly felt, this too was infiltrated. This pain then also disappeared. The patient now was free from pain. The skin thermometer over area *B* now gave a reading of 37·5° C. After two hours the central pain had returned and the thermometer over area *A* gave a reading of 35° C., and that over area *B*, 35·5° C. again.

At operation an inflamed appendix was removed.

We were able to study here both the mid-line pain due to stimulation of the visceral afferents and the iliac pain situated immediately over the site of the inflamed viscus. Both were associated with lowering of the skin temperature and both were abolished by

[1] The hand that is used in palpation must be made warm by vigorous rubbing together of the two hands. It is then passed under the clothes and placed flat on the skin, the whole hand being brought in contact with the skin. The hand is gently lifted and placed flat again repeatedly on different areas of the skin. The "cool" area will then be readily detected when it comes under the centre of the hand.

subcutaneous infiltration with "Novocain". This experiment is therefore submitted as a crucial experiment in support of the hypothesis put forward in this paper.

Patient B (diagnosis, perforated gastric ulcer) complained of a pain in the tip of the right shoulder. On palpation the skin over the painful area appeared cooler than the surrounding skin, and on being tested with the thermometers gave a reading 1° C. below that of the surrounding skin. Similar readings taken on the other shoulder revealed no difference in temperature of one point over another. Infiltration of the "cool" area on the right shoulder abolished the pain.

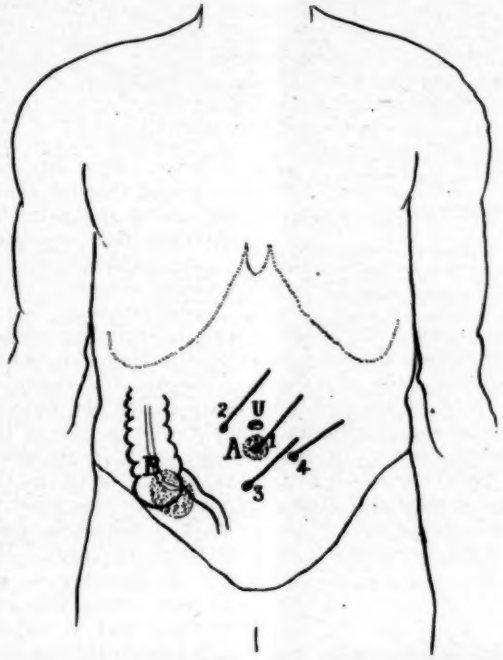

FIGURE VI.

Patient A. See text for explanation. *U* = umbilicus, *A* = vaguely localized mid-line pain, *B* = iliac pain, 1, 2, 3 and 4 = skin thermometers.

We were able to study here phenomena in the shoulder-tip reflexly produced there from stimulation of the diaphragm and giving rise to the sensation of pain. The afferent impulses from the diaphragm pass via the phrenic nerve to the fourth cervical segment. From there they must pass down the cord to the lateral cornu in the upper thoracic cord, where stimulation of the vasomotor neurones gives rise to conditions in the shoulder-tip, which in the case described provided the adequate stimulus for the pain nerve-endings in this region. Pain impulses thus arose there and passed via the pain pathways to the cortex, and the pain was felt in the shoulder because it actually arose there.

Patient C (diagnosis, pleurisy) complained of a sharp pain in the thorax just lateral to and below the right nipple. On palpation a small area could be mapped out which was distinctly cooler to the touch. Skin thermometer readings revealed that this area was 1° C. below the temperature of the surrounding skin. Infiltration of this area with "Novocain" abolished it, but a duller pain was then felt further back.

Stimulation of subpleural somatic afferents reflexly produced phenomena in the overlying tissues which resulted in pain. Weiss and Davis describe numerous experiments of a similar nature in cases of disorder of various viscera, so there is no need to give further cases. Also there is their account of experiments on normal subjects in which localized pain was induced by distending the œsophagus, and the pain was abolished by infiltration over the painful skin area.

Movement of Flatus.

One observation put forward to me as evidence of sensation arising in the viscera is this. During increased peristalsis, particularly in the colon, due to the passage of flatus, the position of the part of the bowel which is locally distended can often be determined, and as the flatus moves along the bowel this movement can also be noticed. The sensation is not painful (unless the distension is excessive, and then it is felt in the mid-line), and it seems to me that the sensations are due to sudden stimulation of the somatic nerves in the parietes. There is ample evidence that ordinary stimuli applied to the parietes can give rise to such sensation, and their occurrence does not therefore appear to effect my hypothesis.

Colicky Pain.

Colicky pain is usually the result of increased tension in the muscle walls of the hollow viscera. Some of these hollow viscera—those derived from the yolk sac, and including the stomach, intestines, gall-bladder and bile ducts—are supplied with sympathetic afferents, and their stimulation gives rise to intense mid-line pain (Morley's true splanchnic pain).

Those organs which are derived embryologically from the intermediate cell mass and belong to the urino-genital system, are probably supplied to some extent with somatic afferents like the rest of the parietal tissue of the abdomen. When these are stimulated, therefore, the pain is definitely localized over the region of the organ (Morley's peritoneal referred type of pain).

Unfortunately I was not given the opportunity of studying a case of renal, intestinal or biliary colic, so that I have no observations to record; but Weiss and Davis report cases in which the pain of renal colic and of gall-bladder origin was abolished by subcutaneous infiltration with "Novocain", so I have no doubt that the mechanism is the same as that described above.

Suggested Explanation of the Localization of Pain.

It has been estimated that the visceral afferents amount to only 2% of the total afferents of the body, and therefore visceral areas are only comparatively scantily innervated compared with somatic areas.

Whereas, therefore, as is well known, there is considerable overlapping of the segmental areas

supplied by somatic afferents, there is probably very little overlapping of the areas supplied by the visceral afferents.

Consequently, in contrast with what occurs in somatic stimulation, it is possible that, when visceral afferents are stimulated, the impulses tend rather to enter a single segment of the cord. Here, owing to the fact that the central processes of the afferent neurones branch and give off ascending and descending collaterals, the impulses enter not only the segment of entry, but also at least those above and below it (see Figure VII).

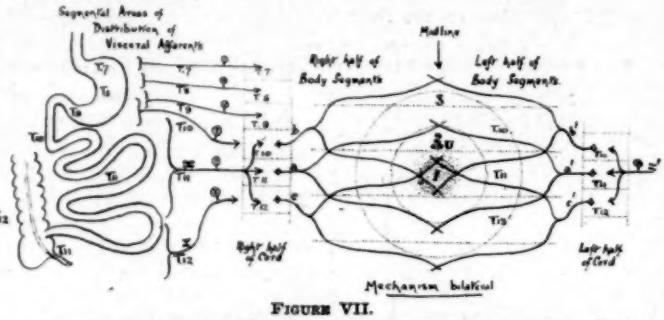
Segmental Areas of Distribution of Visceral Afferents
Right half of Body Segments
Left half of Body Segments
Midline
Right half of Cord
Left half of Cord
Mechanism bilateral

FIGURE VII.

x and x' = the visceral afferent nerves of a particular segment; a and a', b and b' *et cetera* = the vasomotor nerves of a particular segment; 1 = area in which conditions first arise causing sensations which enter the threshold of consciousness as pain sensations.

The visceral afferent impulses enter the both sides of the cord, and consequently vasomotor impulses are discharged from both sides of the cord into the corresponding halves of the body segments concerned. Owing to the central anastomoses of the vessels, both anteriorly and posteriorly, it is in the area marked 1 that the conditions first arise causing sensations which enter the threshold of consciousness as pain sensations. More violent disturbance leads to extension of pain over area 2, and further, area 3 *et cetera*, as more and more segments are involved. These extreme conditions, however, probably occur only in such cases as those of intestinal and biliary colic.

When an inflamed viscus comes in contact with the peritoneal peritoneum, however, this leads to stimulation of somatic afferents, and these only on the one side (see Figure VIII). Now, owing to the overlapping of the peripheral areas of distribution of somatic afferent nerves, the afferent impulses set up reflexes through several adjacent segments. Also, on account of the relative abundance of somatic afferents, the various parts of any individual segment are more exactly represented in the central organ.

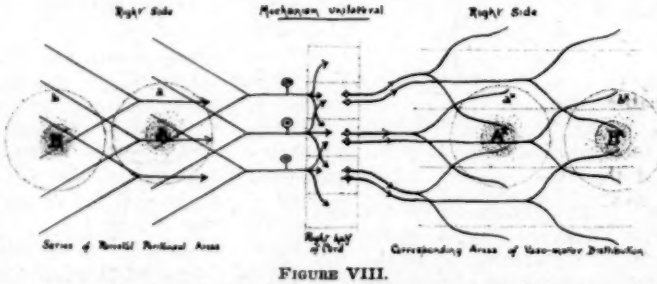

FIGURE VIII.

A (also B) = centre of area of stimulation; A^x (also B^x) = area of maximum vasomotor effects.

In a similar way, the vasomotor nerves probably innervate areas corresponding roughly to the areas of cutaneous distribution of afferent nerves, resulting in maximal effects over a localized area in a particular segment and over the centre of the disturbance.

In the case of visceral stimulation, total segmental vasomotor disturbance first produces in the mid-line conditions giving rise to sensations which enter the threshold of consciousness, while somatic stimulation leads to partial segmental disturbance and produces the same conditions localized over the centre of the disturbance. It seems to me that it is probably a matter of richer central representation of somatic than of visceral areas, permitting more localized results in the former than in the latter.

Use of "Novocain" as an Aid in Diagnosis et cetera.

The fact was mentioned above that when pain was abolished by infiltration with "Novocain" it often appeared in another area, and it was suggested that the abolition of the more intense pain revealed the presence of the less intense. This phenomenon suggests to me a very useful aid in determining, among other things, the more exact localization of a lesion. Take the case of patient A above, for example. Abolition of his mid-line pain revealed the pain in the iliac region situated roughly over the site of the affected viscus. In another case, when a shoulder-tip pain was abolished, pain was revealed in the epigastrium; and Morley reports a reverse case, in which abolition of the mid-line epigastric pain revealed the presence of a shoulder-tip pain.

It appears to me from these observations that in certain cases both the visceral afferents and parietal peritoneal somatic afferents are stimulated simultaneously, and reflex effects are produced not only in the mid-line, but also over the site of the affected viscus. The pain endings are stimulated in both situations, but the patient is conscious of pain in one area only. When this is abolished, however, he is conscious of pain in the other. If, however, the area of parietal peritoneal stimulation happens to be situated just under the area of the mid-line pain, due to stimulation of the visceral afferents, it is obvious that the two pains cannot be differentiated. This is what is likely to occur sometimes in cases of gastric ulcer, and may explain why Morley and Bolton obtained different results in their experiments.

Infiltration with "Novocain" not only brings relief to the patient by abolishing his pain, but it offers a very useful aid to diagnosis.

Reverse Effects.

Pain primarily due to visceral disorders and felt in somatic areas is often relieved by application of heat and counter-irritants. Heat produces local vaso-dilatation (axone-reflex of Langley) and thus probably counteracts the influence of vaso-constriction produced reflexly through the cord from adequate stimulation of the visceral afferents. Counter-irritants may do the same, but it is not improbable that the application of heat and counter-irritants reflexly causes "inhibition" of the gut by excitation of the lateral cornual cells, thus relieving the pain by abolishing the adequate visceral stimuli. Ice-packs also relieve similar pains, but this is probably due to their anæsthetizing effect, thus producing the same effect as infiltration with "Novocain".

Remote Effects from Affected Teeth.

There is in the field of dentistry an interesting application of the theory of the mechanism of visceral pain put forward in this paper. Just as afferent visceral stimuli cause remote vasomotor effects, so afferent stimuli from diseased teeth probably reflexly cause vasomotor effects in the orbit and the sinuses, leading to ophthalmic disorders and disorders of the sinuses. It is well known that disorders of the teeth can cause these remote effects and I make the suggestion that the mechanism is similar to that described above for visceral affections. I have examined cases of toothache and have detected lowering of the skin temperature in the face; and also it is well known how frequently application of warmth to the face relieves the pain.

Summary.

1. From the result of my own observations and those recorded in the literature the following conclusions are reached: (*a*) So-called true visceral pain is due to peripheral effects produced primarily by abnormal stimulation of the visceral afferents. The neural mechanism involved in the production of these effects is the viscero-vasomotor reflex. (*b*) Distinctly localized pain, hyperalgesia and protective rigidity are also sometimes due to the same mechanism, but can also be due to similar effects produced by stimulation of somatic afferents arising in the sensitive parietal tissues of the body cavities.

2. The localization of pain, the possibility of an "irritable focus" in the cord, and the use of "Novocain" as an aid to diagnosis are also discussed.

3. The suggestion is made that remote effects from diseased teeth are probably produced by a similar mechanism.

Conclusion.

In submitting my hypothesis for your consideration, I do it with considerable humility, considering the men whose work has been mentioned this evening. Although for didactic reasons my presentation may have seemed to you a little dogmatic, I am fully conscious of the many questions that remain to be answered. I have merely tried to explain observed phenomena and to arouse your interest in this interesting subject. If I have done this, my purpose is fulfilled.

Acknowledgements.

Before closing, I would like to thank both Dr. Funder, of the Adelaide Hospital, and Dr. Lee, of Brisbane, for the interest they have displayed in these experiments and for supplying useful data. It is difficult for one engaged in an anatomy school situated at some distance from the hospital to get suitable cases for experiment, and it is mainly due to the enthusiasm of these colleagues that sufficient data were obtained to enable me with more confidence to put forward my views tonight. I wish also to thank Professor Sir Stanton Hicks for the loan of skin thermometers. Those I am now using were obtained locally from the firm of Messrs. Wilson and Nafis, who copied those used in Adelaide, which were supplied to them as samples.

References.

(1) A. E. Mills: "Vascular Disturbances as an Explanation of Nervous Symptoms", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1928, page 176.
(2) H. J. Wilkinson: Proceedings of the Medical Sciences Club of South Australia, THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, 1935, page 425.
(3) A. Haller: "Opera Minora", Volume I, 1763.
(4) John Hilton: "Lectures on Rest and Pain", edited by W. H. A. Jacobson, Tenth Edition, 1907.
(5) J. Ross: "On the Segmental Distribution of Sensory Disorders", *Brain*, Volume X, 1887, page 350.
(6) James Mackenzie: "Symptoms and their Interpretation", Fourth Edition, 1920.
(7) K. G. Lennander: *"Über die Sensibilität der Bauchhöhle und über locale und allgemine Anästhesie bei Bruch Operationen"*, *Centralblatt für Chirurgie*, Volume XXVIII, 1901, page 209.
(8) H. Head: "On the Disturbance of Sensation, with Special Reference to the Pain of Visceral Disease", *Brain*, Volume XVI, 1893, page 1; also *Brain*, Volume XVII, 1894, page 339.
(9) H. Head, W. H. R. Rivers and J. Sherren: "The Afferent Nervous System from a New Aspect", *Brain*, Volume XXVIII, 1905, page 99.
(10) A. F. Hurst: "The Goulstonian Lectures on the Sensibility of the Alimentary Canal", 1911.
(11) J. I. Hunter: "Abdominal Pain and its Associated Phenomena", THE MEDICAL JOURNAL OF AUSTRALIA, August 7, 1920, page 115, August 14, 1920, page 143.
(12) A. Lemaire: *"La perception des douleurs viscérales"*, *Revue médicale de Louvain*, Number 6, 1926, page 81, and *"Les douleurs viscérales"*, *ibidem*, Numbers 9 and 10, 1926, page 129.
(13) Soma Weiss and David Davis: "The Significance of the Afferent Impulses from the Skin in the Mechanism of Visceral Pain. Skin Infiltration as a Useful Therapeutic Measure", *The American Journal of the Medical Sciences*, Volume CLXXVI, 1928, page 517.
(14) John Morley: "Abdominal Pain", 1931.
(15) Charles Bolton: "Observations on Referred Pain", *Brain*, Volume LVII, 1934, page 211.

Addendum.

Since this paper was submitted for publication there has come to hand an article by Pollock and Davis,[1] of the Northwestern University, Chicago, in which is put forward a theoretical mechanism of visceral referred pain similar to the above. These authors find that when the intercostal nerve supply is interrupted, either by transection of the spinal cord or by section of a thoracic posterior root, pain produced by stimulating the peritoneal diaphragm by the faradic current can be stopped by severing the phrenic nerve or removing the cervical sympathetic chains; by

[1] Lewis J. Pollick and Loyal Davis: "Visceral and Referred Pain", *Archives of Neurology and Psychiatry*, Volume XXXIV, November, 1935, pages 1041-1054. (In the Publications from the Institute of Neurology, Northwestern University Medical School, Volume VII, 1935.)

severing the eighth cervical and the first, second and third dorsal anterior roots; by severing the cord at the seventh cervical segment; by destroying the cord at the first and second dorsal segments, and by severing the cervical posterior roots. They conclude that the pain produced by faradic stimulation of the peritoneal diaphragm travels over the phrenic nerve; entering the cord by way of the posterior roots, it descends to the level of the eighth cervical and first, second and third thoracic segments; then a connexion is made with cells in the intermediolateral column, and sympathetic efferent impulses travel over the preganglionic fibres through the anterior roots to the cervical sympathetic ganglia. From here postganglionic fibres travel to the skin, blood vessels, meninges and other structures, where, by some vasomotor (?) or hormonal (?) process, the sensory endings of the cerebrospinal system are stimulated and a sensory impulse travels over the ordinary cerebro-spinal system, enters the spinal cord through the posterior roots and ascends to consciousness.

FUNNY TURNS.[1]

By S. O. COWEN, M.D. (Melbourne),
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FOR the title of this address I am indebted to my old friend Dr. F. H. Moran, who, in the somewhat distant days when we were fellow students, propounded in *The Speculum* an ideal examination paper in medicine. His first question was: "Discuss the etiology, diagnosis and treatment of funny turns as seen in the out-patient department." I thought, even then, that the frequency of this strange complaint justified more serious study than my irrepressible colleague had intended. Ten years later, I found it almost as common in private as in hospital practice, and some means of differentiating between seizures indicative of grave organic disease and those which could be safely relegated to the category of funny turns became an immediate practical necessity. The text-books of medicine offered little help, and it was only after a good deal of chastening experience that I evolved a working classification which gave me some confidence in making the distinction. I found, too, that the management and treatment of these aberrant seizures presented problems no less difficult than those of diagnosis.

My interest in the subject, already keen, was quickened by reading, in 1934, a paper by J. A. Ryle on "Some Alarming Seizures", which set out, much more ably than I can hope to do, conclusions somewhat similar to my own. This address has recently been republished in "The Natural History of Disease", which is, by reason of the soundness of its matter, no less than the excellence of its style, one of the outstanding medical works of our time. I had hardly realized, when I undertook the preparation of this address, how closely it would follow Ryle's work. He has covered the ground so adequately and expressed his conclusions so clearly that it is difficult to avoid repetition of his actual words. I feel that I can escape a charge of plagiarism only by freely acknowledging my indebtedness to his paper.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on August 29, 1936.

Definition.

Funny turns may be defined as transient attacks, often accompanied by distressing or even alarming sensations and sometimes by loss of consciousness, which, though they may occur in patients who are suffering from organic disease, are not themselves dependent on gross structural lesions in the brain, heart or other organs.

Epilepsy, both major and minor, falls within the scope of this definition, but though its seizures are often described by the patient as funny turns, their serious import does not permit the physician so to regard them. Paroxysmal tachycardia is so obviously a cardiac disorder and is so seldom accompanied by symptoms referable to other organs that its attacks, though they conform strictly to the terms of our definition, are usually referred to as "heart turns". I propose, therefore, to omit these two and to confine my remarks to migraine, paroxysmal labyrinthine vertigo, fainting and vaso-vagal attacks and the paroxysmal manifestations of abdominal allergy. In discussing them, I shall allude to that most prolific cause of funny turns, the anxiety state.

Migraine.

Although the typical migrainous seizure, with its prostrating headache, preceded by visual phenomena and accompanied by nausea and vomiting, is usually described by its victim as a sick headache, its incomplete and aberrant forms are amongst the commonest causes of the funny turns encountered in general medical practice.

Incomplete Migraine.

In some migrainous subjects the attacks do not always assume the classical form. Patients who suffer from typical migraine in early life, may, in later years, experience only transient headaches unaccompanied by visual disturbance or vomiting. In others, attacks which stop short after the appearance of the prodromal symptoms may alternate with fully developed seizures. Incomplete attacks such as these are almost always described by the patient as funny turns, and unless the history is carefully scrutinized, their true significance may be missed. On the other hand, it must be recognized that anomalous recurrent seizures may arise from other causes. Confusion will be avoided if we ascribe to migraine only those transient paroxysmal disturbances which occur periodically in persons who have been at some period of their lives subject to more typical attacks presenting at least two of the three characteristic symptoms: teichopsia, headache and vomiting.

Though the cerebral disturbance which ushers in the attack of migraine is usually confined to the occipital lobes, it may affect any part of the brain. In 60% of a consecutive series of 50 cases in my own practice the prodromata were visual. The classical fortification spectra were seldom described; more often there were partial scotomata, flashing lights or general diminution of visual acuity. Premonitory symptoms resulting from disturbance of other parts of the brain were described by 15% of

the patients. Of these, transient hemiplegia, aphasia or vertigo and oculomotor paresis of longer duration were the most striking. In some of the patients many attacks stopped short with the appearance of these symptoms and the typical headache failed to develop. In such cases careful questioning revealed the occurrence, at some time or other, of complete attacks of migraine which gave the clue to the correct diagnosis.

CASE I.—*Migraine Variants Masquerading as Funny Turns*: A man of fifty-three years of age, engaged in responsible and exacting work, consulted me in great perturbation because of a funny turn he had suffered earlier in the day. While reading the newspaper during the train journey from his suburban home to his office, he had suddenly become aware that his field of vision was so narrow that he could see only one word of the print at a time; within a few minutes white zig-zag figures had almost completely obscured the whole field. By the time he had reached town, twenty minutes later, the attack had subsided, leaving him perfectly well, though anxious lest some serious visual calamity had befallen him. Inquiry revealed the fact that from boyhood up till the age of forty he had suffered from periodic paroxysmal headaches preceded by brief visual phenomena consisting of bright lights and zig-zags and terminating in bilious vomiting. Since the headaches ceased he had at times experienced peculiar brief attacks of disorientation in which he felt as if the compass had rotated through an angle of 45°, and which ended as a rule when he reached any place the exact situation of which he knew well. He was completely reassured by the explanation that his funny turns were migraine variants and, though he subsequently had several brief attacks of disturbance of vision, they ceased to cause any alarm.

In several of my patients transient aphasia has similarly occurred as an equivalent of the more complete seizures experienced at other times.

Aberrant Migraine.

By aberrant migrainous attacks is meant those that are characterized by unusual manifestations as well as more typical seizures in which one or other of the common symptoms so dominates the clinical picture that the true nature of the complaint may not be recognized.

Vertigo, almost as severe as that which occurs in Ménière's disease, may be of migrainous origin, but as it is commonly encountered in patients in the fifth and sixth decades who have long been subject to more typical attacks, its true nature is usually apparent. The paralytic accompaniments of migraine, as well as being alarming to the patient, are a source of great diagnostic anxiety to the physician.

CASE II.—*Hemiplegic Migraine*: In 1926 I was summoned hurriedly by a woman, aged fifty-seven years, who presented the classical signs of an almost complete left-sided hemiplegia. Because the paralysis appeared at the height of a particularly severe bilious headache, a condition to which she had long been subject, I hazarded the opinion, with much trepidation, that the condition was one of hemiplegic migraine. Her complete recovery, within a few hours, justified the diagnosis. Although she has had several similar attacks since, she is well and active at the age of sixty-seven.

I have seen only one instance of ophthalmoplegic migraine. The diagnosis was again established by the occurrence of the paralysis during an otherwise typical attack. As is usual in these cases, the condition persisted in severe form for many weeks and left behind it some permanent dilatation of the affected pupil. These paralytic manifestations serve as a reminder that migraine, especially when it arises in later life, is sometimes symptomatic of organic cerebral disease.

Even when migraine occurs in its fully developed form, one of the cardinal symptoms may assume such importance in the history that the others are apt to be overlooked. This applies especially to nausea and vomiting. The patient is often so convinced of the bilious origin of his trouble that he ascribes each attack to some article of food ingested just before its occurrence. Sometimes this attitude of mind leads to unwarranted restriction of essential foodstuffs with consequent impairment of general health and increased frequency of attacks. In cases of this sort the dietetic aspect of the history bulks so largely that the examiner may fail to recognize the migrainous origin of the complaint.

CASE III.—*"Bilious Turns" due to Migraine*: A male patient of twenty-seven years complained that during the previous five years he had been subject to frequent and severe bilious attacks. They started suddenly with alternate chills and flushes, followed within a few minutes by sweating and by intense giddiness so that he walked just like a drunken man. With the onset of the vertigo his vision was affected in such fashion that perpendicular lines, as of walls of buildings, appeared to be "off the straight". Vomiting usually occurred within two hours of the onset of the attack and brought instant relief from the more severe symptoms, but slight nausea and giddiness persisted until he had had a sleep. Only on direct questioning was the fact elicited that in some attacks generalized headache had occurred. Because of the prominence of vomiting he had regarded the seizures as "bilious turns". As the first one had occurred soon after a breakfast of boiled eggs, he had rigorously excluded eggs from his diet. In the same way he had incriminated and subsequently abstained from meat, fats and fried fish, restricting himself almost entirely to starchy foods. As a result he had lost over a stone in weight, and with the decline in his general health the attacks had become more frequent. Explanation of the nature of the attacks and reassurance as to their innocuousness, together with mild sedation and restoration of an adequate dietary, so improved matters that the attacks ceased to cause any trouble.

The Treatment of Migraine.

While it is true that funny turns, apparently of bilious origin, are often really migrainous, it is also quite certain that in some patients dietetic indiscretions may provoke attacks of migraine. Careful inquiry should be made whether the seizures may reasonably be ascribed to any particular article of food. Chocolates, eggs, cream and meat in excess are the worst offenders, and their exclusion from the dietary sometimes diminishes the number and severity of the attacks. Alimentary toxæmia is, however, much less potent as a precipitating cause than are nervous stimuli, such as excitement and, especially, fatigue. In women these nervous factors are particularly prone to provoke attacks at or before the menstrual periods. This fact and the frequency with which the seizures change their character at the menopause suggest that endocrine activity plays an important part in the ætiology of migraine. But, so far, this suggestion has not led to therapeutic methods of any

value. Sedatives such as bromide and "Luminal", given in small doses over long periods, are the most useful drugs in the treatment of migraine. They damp down the sensitiveness to nervous stimuli which characterizes the migrainous diathesis and so lessen the effect of fatigue and of emotional disturbances. The attack itself may often be aborted by the hypodermic injection of half a cubic centimetre of ergotamine tartrate. But what is most needed by the sufferer from funny turns of migrainous origin is a full explanation of the nature of his seizures and reassurance as to their innocuousness.

Vertigo.

I do not propose to attempt a scientific consideration of vertigo, a task which is beyond the scope of this address; my intention is rather to outline my own summary classification of cases in which the occurrence of attacks of giddiness is the presenting symptom. Although it is neither comprehensive nor scientifically exact, it has proved useful in distinguishing between those types of vertigo which betoken organic disease and those which may safely be regarded and treated as funny turns.

In the first place I make a distinction between dizziness and vertigo which, though it may be etymologically indefensible, divides patients who suffer from any sort of giddiness into two large groups. By dizziness is meant a feeling of insecurity and loss of balance not accompanied by apparent rotatory movement of the body or of external objects; it is often described by patients as "light-headedness". It may occur in association with arteriosclerosis and other organic diseases, but it is much more commonly a manifestation of functional disturbances, such as vaso-vagal and fainting attacks, the effort syndrome and the anxiety state. The term vertigo is restricted to loss of equilibrium associated with apparent rotation of the body or of external objects. It may be symptomatic or labyrinthine or cerebellar disease, in which case it is usually persistent. On the other hand, it may arise from purely functional disturbance of the vestibular mechanism, when it is more likely to be intermittent and paroxysmal. Whatever its origin and duration, vertigo, as here defined, causes much more distress than does dizziness.

Apart from the vertiginous form of migraine, which has already been briefly discussed, the causes of vertigo can be grouped under five headings. These groups, together with their relative incidence in a consecutive series of fifty private patients in whom vertigo was the presenting symptom, are as follows:

1. Gross intracranial disease accounted for 6% of the cases. The vertigo was usually continuous and abnormal neurological signs were persistently present.

2. Nine *per centum* of the patients suffered from chronic *otitis media*.

3. In 16% of this series the vertigo, which was of sudden onset and great intensity, persisted without intermission for periods varying from a few days to three weeks. While it was at its height bilateral signs of cerebellar disease could usually be elicited. Tinnitus was invariably present in both ears as long as the vertigo continued. This syndrome may be due to acute vestibulitis, but it differs from the text-book description in that complete recovery has occurred in all my cases.

4. Although true vertigo is seldom a neurotic manifestation, it was encountered as the dominant symptom of an anxiety state in 8% of this group of patients. Because anxiety and apprehension so often accompany vertigo due to other causes, no patient should be relegated to this category without the most careful consideration.

5. Paroxysmal labyrinthine vertigo was diagnosed in 61% of the cases. This term is preferable to Ménière's disease, because the transitory character of the vertiginous attacks makes it obvious that they are not the result of labyrinthine hæmorrhages, as originally suggested by Ménière. The most recent hypothesis is that they are produced by variations in the tension of the endolymph. But whatever their pathological basis, it is vertiginous seizures of this kind which especially merit the description of funny turns.

Paroxysmal Labyrinthine Vertigo.

The typical attack of paroxysmal labyrinthine vertigo is generally easy of recognition. A patient who is the subject of persistent tinnitus and slowly increasing deafness is suddenly smitten down by calamitous vertigo. The attack is brief and is accompanied by, at most, only transient loss of consciousness. Apart from the tinnitus and deafness, the patient suffers no disability between the seizures, which eventually cease when the deafness becomes complete. Nausea and vomiting are not constant features of the attacks, but sometimes they bulk so largely in the patient's recital of his symptoms that the concomitant giddiness, which gives the clue to the correct diagnosis, is disclosed only by careful questioning. Pallor, sweating and collapse, with some degree of bradycardia, always accompany the vertigo; occasionally they are so obtrusive that the attacks are wrongly ascribed to heart block or some other cardiac disorder. The following case illustrates this point very clearly:

CASE IV.—*Paroxysmal Labyrinthine Vertigo Simulating Stokes-Adams Disease*: An over-worked medical man, aged forty-seven years, engaged in a busy suburban practice, was referred to me because of alarming attacks which his own doctor regarded as due to heart block. In 1916 he was invalided from the army because of severe bronchitis accompanied by asthma. The asthmatic attacks were frequent and severe, but they steadily lessened in number and intensity, and by 1926 they had almost ceased. In 1927, however, he began to suffer from giddy attacks with vomiting; they were mild in character, occurred about once in every two months, and caused no real incapacity. He was deaf in the left ear as a result of middle ear disease in childhood, but there was no tinnitus. During 1932 he was particularly busy and was much bothered by family cares. On January 4, 1933, he had a calamitous attack of vertigo which seriously shook his confidence. Other severe seizures followed and his general health greatly deteriorated. His wife's description of the attacks was as follows:

First of all, the attacks always come on when he is definitely over-tired, either through over-work or some little extra physical exertion. Sometimes it is just giddiness and a "muddly" feeling in the head, but in all the severe attacks there is a definite kind of routine about it. First he becomes suddenly very giddy and fairly staggers to the nearest chair or couch. The excessive giddiness, which continues all the time, even when the eyes are closed, causes a feeling of nausea, and then he begins violently retching and vomiting. This latter seems to be the cause of the severe abdominal pains which follow, and then the heart collapses. His face goes a bluey-white colour and he comes out in an excessive perspiration all over. Everything goes "black" in front of him, and he always says: "Let me hold onto your hands. I feel I am falling through space." After the severity of the attack is over he suddenly falls asleep in sheer exhaustion and wakes a few hours later, feeling rather shaky, but otherwise wonderfully recovered.

His doctor wrote:

On January 4, 1933, he had an acute collapse, when he must have been very near death. When seen an hour after the attack began he was blanched and sweating profusely, vomiting occasionally; heart rate 48, doubtless owing to heart block.

Several attacks of comparable severity followed, and when I saw the patient in September, 1933, he was an anxious, nervous wreck and quite incapable of doing his work. Apart from the evidences of his old *otitis media*, with some degree of Eustachian block, his physical state, as determined by clinical, radiological and electrocardiographic examinations, was quite normal. The attacks were ascribed to paroxysmal labyrinthine vertigo, and their nature was fully explained to the patient, who was at the same time reassured about his heart condition. "Luminal" and bromide were prescribed and Mr. John Shaw carried out the necessary aural treatment. The seizures forthwith lessened in severity, and by September, 1934, the patient was back at work, although he was still apprehensive about the significance of his attacks and fearful of their recurrence. His confidence and mental poise gradually returned, and in April, 1935, he reported:

I still have occasional attacks, which are brief, but severe. I recover from them very quickly and go on with my work as if nothing had happened. I really feel very well and the attacks do not bother me.

This case illustrates clearly the means whereby vertiginous seizures may be robbed of most of their terrors and relegated to the category of funny turns. Because we do not fully understand the pathology of paroxysmal labyrinthine vertigo, we cannot treat it radically. We can, however, teach our patients to avoid extreme degrees of emotion and fatigue or other external agencies which the history may incriminate as exciting causes of the attacks. We can, too, see to it that the condition is not aggravated by inflammatory processes in the ear or by toxæmia arising from infections elsewhere in the body. But, above all, we can deal with the most significant perpetuating cause of the attacks: the anxiety and apprehension engendered by the seizures themselves. Ryle puts the importance of this factor very clearly:

Nothing could be better devised to provoke nervous instability than attacks of genuine physical instability coming on without warning and beyond all voluntary control.

Once the patient can be brought to realize that the vertiginous episodes are transient and paroxysmal and not significant of organic cerebral or cardiac disease and that he can lead a life of almost normal activity between the attacks, he will lose most of his anxiety and the seizures will lessen in number and severity. Phenobarbitone and bromide, alone or in combination, help to allay apprehension and damp down nervous irritability, but explanation and reassurance are the essential features of the treatment of paroxysmal labyrinthine vertigo.

Fainting and Vaso-Vagal Attacks.

A mild faint is often described as a funny turn, and its nature can usually be determined from the patient's description of his sensations during the attack. The fact that sensory disturbances, apparently of visceral origin, may accompany a faint should always be borne in mind. They are commonly of brief duration and are described as feelings of "going away" or "falling". In their most severe form they give rise to the terrifying sense of impending dissolution which characterizes the syndrome described by Gowers as the vaso-vagal attack, and by German authors as *angina vasomotoria*. Attacks of this kind may last for hours and may be accompanied by cardiac pain and signs of collapse. The more severe forms have, fortunately, occurred but rarely in my practice. Like the simpler fainting attacks, they are provoked by emotion and exhaustion or by the close atmosphere of crowded rooms; and though they often occur in patients who are debilitated by physical illness or mental stress, they are not directly due to cardiac or other organic disease. The treatment of these syncopal attacks, from the mildest to the most severe forms, is along the same lines as that of paroxysmal vertigo, and is attended with similar satisfactory results.

Abdominal Allergy.

Under the heading of abdominal allergy I would tentatively class three instances of sudden and alarming seizures which patients have described as funny turns. Their characteristics may be exemplified by a case history.

CASE V.—A gentleman, eighty-nine years of age, who is hale and hearty and in full possession of all his faculties, has "all his life" been subject to "collapse turns". These are heralded by a queer, uneasy, epigastric sensation, which, he says, cannot be described either as indigestion or as pain. Within an hour or so after its onset he becomes suddenly faint, his skin acts slightly and he feels an urgent desire to go to stool. Two or more copious watery motions are passed at intervals of about thirty minutes. Vomiting occurs in some of the attacks, but neither headache nor visual phenomena have ever been noticed. He feels tired and languid for some time after the cessation of the diarrhœa, but within four or five hours of the onset of the attack he has completely recovered and is able to consume a hearty meal.

Attacks such as these would be classified by some authorities as abdominal migraine, but the absence of headache, in my opinion, excludes them from that category. The term "abdominal allergic attacks" will, I know, be challenged, but in using it I have the support not only of Ryle, but also of Hurst, who has laid down criteria for the recognition of allergic attacks somewhat analogous to Koch's postulates. Though I cannot claim that they have all been satisfied in my own cases, I continue

to regard attacks of this kind as allergic, because by doing so their transient and innocuous nature is emphasized. Their general treatment must be on the same lines as that of migraine, but, in addition, I have found ephedrine of some use as a prophylactic. The attacks are so sudden that no opportunity has presented itself of using adrenaline as a means of aborting them; if it were successful, it would, I think, finally establish their true nature.

Summary.

Many of the funny turns encountered in general medical practice are of neurotic origin. Others are due to the commoner paroxysmal neuroses: migraine, paroxysmal labyrinthine vertigo and vasovagal attacks. Some few may be allergic.

Attacks due to any of these causes may produce undue alarm in the minds of both doctor and patient, but recognition of their true significance permits their relegation to the category of funny turns.

The essentials in the treatment are explanation and reassurance. Sedative drugs occupy a useful but subsidiary place.

Bibliography.

J. A. Ryle: "Some Alarming Seizures", *The British Medical Journal*, Volume I, 1934, page 89; "The Natural History of Disease", 1936.

A. F. Hurst: "Gastro-Intestinal Allergy", in Price's Text-Book of Medicine, Fourth Edition, 1933.

Reviews.

EXOPHTHALMIC GOITRE.

THE second edition of Israel Bram's "Exophthalmic Goitre and its Medical Treatment" should be of great interest to all those concerned with the problems of the treatment of this condition, for Dr. Bram is evidently firmly convinced of the adequacy of proper medical treatment, and his published results are amazingly good.[1]

His contention is that, "strictly speaking, exophthalmic goitre or Graves's disease is not goitre; the malady presents widespread implications etiologically, clinically and therapeutically, and should be classified under constitutional conditions". The method adopted in the writing of the book has accordingly been such as would stress the importance of constitutional factors and the urgent necessity for the intensive study of the individual, particularly from the psychological aspect.

The nature of the disease, ætiology, symptomatology, physical signs, diagnosis and differential diagnosis are fully discussed; but we are not satisfied with the chapter, or rather subsection of a chapter, dealing with the pathology of the condition, as the descriptions and discussion are quite inadequate and disproportionately small. The condition of the thyreoid gland in Graves's disease is stated to be specific and pathognomonic, while "toxic adenoma is self-explanatory, indicating the superimposition of a state of thyroid toxicity upon an old-standing tumour of this organ". Pathologists have shown that true adenomata are very rare, while nodular goitre is common; and few physicians will agree that it is a relatively simple matter to differentiate sharply between the two varieties of thyreotoxicosis described by Plummer, and stressed by Bram, although the latter describes twenty points of difference and maintains that "in toxic adenoma the thyroid makes the body ill" and "in exophthalmic goitre the body makes the thyroid ill".

Bram's quinine tolerance test is a very useful addition to the tests which may be employed in determining the presence of thyreotoxicity, and the discussion of other laboratory tests is excellent; but the chief interest of the book is centred in the course, prognosis, mortality (which is only 0·3%), treatment, and end results.

The treatment embraces prophylactic, dietetic, hygienic, medicinal, psychotherapeutic and other measures, based on the assumption that Graves's disease is due to a widespread dysfunction of the endocrine glands and of the autonomic system, and "is not treatment for the lump in the neck". Thyreoidectomy is considered to be necessary in only slightly more than 2% of cases, and then chiefly for pressure manifestations, and is consequently dealt with very briefly; but the details of medical treatment are fully and lucidly described.

The end results in 2,600 patients followed up by personal contact for varying periods of from three to twenty years show 90% to be entirely well. Of the remainder, most had come under observation after thyreoidectomy or irradiation of the thyreoid, and even in these the disabilities were slight. These results alone challenge surgeons, radiologists, physicians and general practitioners to review their respective positions regarding the treatment of exophthalmic goitre; they will readily overlook the frequent unnecessary repetition which occurs, and will not regret the time spent in careful perusal of this book, which is well printed and contains ample references.

Dr. Bram is to be congratulated on his results, and his book should attain its main purpose, which is "to clarify and amplify the definition of medical treatment and assist in guiding patients back to enduring health and happiness".

[1] "Exophthalmic Goiter and its Medical Treatment", by I. Bram, M.D., with foreword by R. G. Hoskins, Ph.D., M.D.; Second Edition; 1936. St. Louis: The C. V. Mosby Company; Melbourne: W. Ramsay. Royal 8vo, pp. 456, with 79 illustrations. Price: 36s. net.

LABORATORY METHODS.

"A SYNOPSIS OF CLINICAL LABORATORY METHODS", by Dr. W. E. Bray, is a small and extremely compact volume.[1] The author's aim, as stated in his preface, is to collect in one volume all the most frequently used methods of laboratory diagnosis. He includes all branches of pathological work and laboratory diagnosis preparation in a small volume of 300 pages. The type is small but clear, the style is extremely condensed, dogmatic and business-like, reminding one irresistibly of a first-class cookery book. Like a good cookery book, this volume contains much useful information and many good and tried recipes, but it also describes several methods which the author has quite obviously never tried, or at least which could not be successfully carried out from his inadequate description of them.

The directions for the performance of the simpler laboratory procedures contain many useful and practical details that mark the author as a careful and experienced worker. But the technique of such a complicated matter as anaerobic culture methods, for instance, is described in half a page, just as though it were as simple as pricking the finger to obtain blood. Such anomalies and unevennesses make a book of this kind misleading to an inexperienced worker, while the more experienced person will prefer to consult books of a better type, monographs, or original articles.

The book is quite generously illustrated, and some of the illustrations are excellent. Others, notably the pictures of the cells of the blood and of malarial parasites, are very poor indeed. It is an heroic task nowadays to attempt to include directions for the work of a bacteriologist, a morbid anatomist, and a biological chemist in one small volume. Bray includes not only directions for laboratory tests, but also indications for them, generously throwing in such extras as a classification of nephritis, an account of the anæmias, a list of the indications for metabolism estimations, and so on. These interpolations are surely out of place and must of necessity be superficial.

[1] "Synopsis of Clinical Laboratory Methods", by W. E. Bray, B.A., M.D.; 1936. St. Louis: The C. V. Mosby Company. Crown 8vo, pp. 324, with 32 illustrations and 11 colour plates. Price: $3.75 net.

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SYPHILIS.

NEARLY eighteen months ago the treatment of syphilis was discussed in these pages in the light of a report by Professor Hans Martenstein, published in the *Quarterly Bulletin of the Health Organization* of the League of Nations. It will be remembered that Martenstein's report was prepared because of the unsatisfactory therapeutic results obtained in certain places and with the object of laying down a plan for uniform and continuous treatment. Martenstein's investigations revealed a loose standard of diagnosis and treatment; he found that in some places methods were adopted that could not possibly be satisfactory. The obvious conclusion was that if careful use of available methods was made, better results might be expected. John H. Stokes, who, as Duhring Professor of Dermatology and Syphilology of the University of Pennsylvania, is striving for the control of syphilis, has adopted a somewhat different method of arousing interest. In his Prosser White Oration, delivered before the St. John's Hospital Dermatological Society, he has attacked what he calls the rising tone of unguarded optimism among even the most experienced, for "uncritical over-confidence defeats itself".

Many will question Stokes's view that unguarded optimism is prevalent even amongst experienced campaigners, that we picture ourselves "equipped to all outward appearances with such drugs, such methods, such prospective organization, such command of critical strategic points like the control of infectiousness, that a proclamation emphasizing the hopeful and the possible reads like a medical fairy tale". To under-estimate a foe or to ignore his capabilities is foolish; it may therefore be well to consider some of the views that Stokes puts forward. It is, of course, true that the characteristic viability, virulence and infectivity of the *Spirochæta pallida* have prevented universal infection of the human race; but we need to be reminded that it is an organism peculiarly suited to its human habitat in what has been called the most exquisite example of symbiosis in the pathogenic field. In time no doubt more will be learned of the biological characters of the spirochæte and of the conditions under which it lives and moves and has its being. Stokes describes it as "a parasitic diplomat of extraordinary adaptability, an opportunist whose persistence in the anergic mouse now warns us of the human asymptomatic carrier, and is beginning to arouse the misgivings of both laboratorians and clinicians". He thinks that the anergic, hypergic and hyperallergic states of tuberculosis are beginning to find parallels in syphilis. Refractoriness to treatment is a case in point. We must agree with Stokes that the problems of the spontaneously anergic and the treatment-refractory, together with the problem of the treatment-resistant strain of the organism, are a challenge to present-day chemotherapy. At the same time the point is more of academic than of practical importance. Should refractoriness to treatment become more common (and some observers hold that it is increasing), this statement would have to be revised. On this occasion we wish to lay stress on the practical side of the problem involved in the control of syphilis. We shall therefore merely refer to Stokes's remarks on the

[1] *The British Journal of Dermatology and Syphilis*, November, 1936.

avirulence of the *Spirochæta pallida* on cultivation, to his suggestion that the spirochætal form seen clinically may be only a concomitant of a missing something else, and to the possibility that we may "find ourselves tossed into the lap of the virus diseases as we develop our campaign for control". Reflection on the gaps in knowledge is valuable if it makes us careful in the interpretation of doubtful results and eager to fill in the lacunæ. It may be true that in our fight against syphilis we are relying on "a chemotherapeutic empiricism, not a biochemical rationalism"; but it is none the less true that if we make full use of available methods, we shall not be on the losing side.

This brings us to the question of propaganda and prophylaxis. In dealing with the prevention of syphilis (or any other venereal disease) we come into conflict with the sexual urge, one of the most powerful factors in human life. Of this urge Stokes writes that it can make all popular and medical knowledge of the biology of syphilis and its transmission as meaningless as Chinese script. "With a condom in his pocket, a doctor, and no fool at that, will drink himself into an unprotected folly and come to me the morning after in remorseful panic." And he asks: "What has learning meant to this man, and what can he make it mean to others?" There is a "tragic separateness" of the emotions and the intelligence which is "the *bête noire* of the mental hygienists". Prophylaxis as commonly discussed is essentially weak because it ordinarily follows instead of anticipating the risk; "it demands either the pressure of fear and an iron hand, or three graces rarely present at the same moment on the scene of sexual exposure—intelligence, promptitude, sobriety". The present state of affairs is full of difficulty. Good influences are expanding and permeating the community; but, as Stokes points out, pornography, lubricity and all the forms of literary and cinematic frankness and overstimulation are not giving ground. "Taboo is breaking down, but inhibitions are going with it." The end result is hard to predict. The medical profession should seize every opportunity of spreading accurate knowledge of syphilis and of other venereal diseases, but it should also insist, especially to the young, that self-control is an ideal to be sought, that liberty easily lapses into licence, and that the widespread preaching of prophylaxis is a confession of failure.

Current Comment.

A NEW METHOD OF TREATMENT IN HÆMOPHILIA.

HÆMOPHILIA in all degrees is a curious disorder which at once commands both the dread and interest of physician, surgeon and obstetrician. More than once it can claim to have changed the course of history, and ruined the happiness of many a family. The lives of hæmophiliacs are recorded as having been saved by measures so diverse as a blood transfusion, the use of liver or ovarian extract, and recently the application of viper venom. No method of prophylaxis, however, has ever established itself as of any reliable and constant value.

The accidental discovery that a product of the incubation of egg white and potassium bromide, when added to blood *in vitro*, produced a clear structureless and non-retractile gel, led W. A. Timperley, A. E. Naish and G. A. Clark to try its effect upon the clotting time of cats.[1] It was found to be completely non-toxic even when injected intravenously, and, more important, reduced the coagulation time of the blood appreciably. Timperley repeated the injection upon himself with similar conclusions. Further methods of preparation have been evolved after considerable trial, and the final precipitate dissolved in normal potassium chloride has received a trial in the treatment of persons suffering from hæmophilia. One great difficulty has been that of standardization, since so many varying factors can affect the clotting time. The chief test of efficiency is the effect on the clotting time of a hæmophiliac who has not had a recent dose. It is impossible at present to give any definite indication of the dose which may be anticipated to render a patient free from hæmarthrosis or protect a patient from external hæmorrhage. The spontaneous fluctuations of the disease itself cannot be legislated for, and a dose which by its results may be commended as adequate at one period, may be quite insufficient at another. Apparently there is no tendency towards intravascular clotting, however large the dose, so that for therapeutic purposes a generous dosage is desirable. A tendency to rebleed may perhaps be forecasted by serial regular estimations of clotting time. Following an injection of this new extract, the clotting time rises at first slowly and then more rapidly.

The authors are very conservative in their claims, and point out the difficulty of making any final assessment from the small series of 13 patients reported in their article. They have been prepared,

[1] *The Lancet*, November 14, 1936.

nevertheless, to put their therapy to the test of instructing patients to forget the habitual ingrained caution, and to indulge in muscular exercise and sport which formerly always ended in joint effusion. More than this, they have deliberately performed the dreaded extraction of defective teeth. In eleven tooth extractions on four patients no wool or plug of any description was used. Such operations have given valuable opportunity for the comparative estimation of the effects of varying doses and routes of administration. The dose necessary to check dental oozing is much larger than that required to diminish hæmarthrosis, and needs more frequent repetition. The hæmatoma following puncture of muscle or vein in the course of injections becomes progressively smaller and finally fails to make an appearance. One practical point became evident during the course of this valuable investigation, namely, the necessity for large stocks of injection material to be at hand, because of the rapidity with which the bleeding state may return in some patients. For this reason dental extractions were performed early in the morning, so that close observation by daylight could anticipate the time for the next administration. It appears also that mucic acid is closely related to the substance responsible for this happy result upon blood coagulation. No doubt work is already proceeding in many centres to determine, if possible, some method of identifying and concentrating the responsible substance in the hope that at last some faithful means may be found to succour the fragile hæmophilic community.

RECENT CONCLUSIONS CONCERNING MANDELIC ACID.

How mandelic acid and its salts came to find a high place among the urinary antiseptics of the present time has been recently described in the pages of this journal. A conception is now growing of the exact type of case in which success may be fairly confidently anticipated, and, conversely, when mandelic acid may not lay claim to any superiority over its older competitors.

M. L. Rosenheim has brought forward a further series of "unselected" patients, 88 in number, who have received a syrup of ammonium mandelate in order to cure their urinary infection.[1] He prefaces his remarks by a denial that ammonium mandelate is often a renal irritant, and recalls that Hollingworth and Platt have actually reported favourably on its use in patients with acute nephritis associated with infected urine. The present method of treatment is to give three grammes of mandelic acid in the form of syrup of ammonium mandelate four times daily, after meals, and to limit the fluid intake to two pints a day. Before classing any patient as having failed to respond to the drug, it is essential to make sure that the pH of the urine is 5·3 or less, by the simple methyl red test. In some patients it is necessary to supplement the acidifying powers of ammonium mandelate by a further addition of ammonium chloride. In others, infection of the urine by a urea-splitting organism or the presence of renal efficiency rendering impossible the secretion of a sufficiently acid urine is a reason for non-success. Indeed, renal failure is a definite contraindication to mandelate therapy, as any increase in the acid ions in the plasma is undesirable. In the series of patients here reported, 71% of apparent simple infections were cured. In acute pyelitis or cystitis alkalis must be given a thorough trial first. A *régime* of restricted fluid intake, with the production of a highly acid urine, has obvious disadvantages in the presence of an acutely inflamed renal tract.

Sterilization of the frequent bacilluria remaining after the alkali therapy by mandelic acid is both desirable and efficacious, particularly from the point of view of the prevention of future relapse. Mandelate therapy has achieved its most outstanding successes in the cure of patients with long-standing infection by the coliform organisms. Failure is fairly common, however, when urinary stasis results from mechanical obstruction of a surgical nature. It is claimed, however, that even though sterilization prove difficult, symptomatic relief and diminution in the degree of pus formation are frequently achieved. A "test" treatment by adequate mandelate administration may save the discomfort and expense of a full instrumental investigation. If this fails, a complete urinary examination becomes indicated.

The exasperating condition known as pyelitis of pregnancy usually fails to respond to mandelic acid any more than to any other urinary antiseptic, but considerable symptomatic relief is claimed by Rosenheim. *Bacillus proteus* infections are also resistant. In tuberculous pyelitis an associated infection with secondary bacterial invaders may be expected to disappear. In chronic cystitis oral therapy with mandelic acid can be combined with advantage with local lavage with a 2% solution of the same substance at pH 5·0. The criterion of cure is a sterile urine culture taken several days after the cessation of treatment. In spite of this, relapses are common. To combat them it is better to take the full dose of acid for a few days once a fortnight than gradually to reduce the dose and so to lose effective concentration. The main contraindication remains that of a high blood urea content.

It appears, therefore, that ammonium mandelate will be the fashionable antiseptic for some time to come. It is not unlikely that it will be replaced by another introduced with a similar enthusiasm. One speaker at a discussion on this subject at a meeting of the Royal Society of Medicine, wondered whether hexamine would not be just as satisfactory if similar care were taken in its administration, and in particular in the associated exact control of urinary acidity.

[1] *The Lancet*, November 7, 1936.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Hypertensive Fundus Oculi after Resection of the Splanchnic Sympathetic Nerves.

F. B. FRALICK AND M. M. PEET (*Archives of Ophthalmology*, May, 1936) relate their experience with the operation of resection of the splanchnic nerves in the treatment of angiospastic retinitis and its allied systemic conditions. The operation consists in the resection of the greater and lesser splanchnic nerves and of the lower dorsal sympathetic chain through a subdiaphragmatic approach, after resection of a portion of the eleventh rib. The operation is performed bilaterally. This operation has now been performed in over 90 patients, all with a systolic blood pressure of 200 millimetres of mercury or more. In 42 cases sufficient time has elapsed to draw conclusions. The operative mortality was 4·8%; no improvement occurred in 26%; there was moderate improvement in 24%; marked improvement in 19%, and apparent cure in 14·4%. It is imperative to recognize arterial constriction or angiospasm as the precursor of angiospastic retinitis. The three objects of operative intervention are the relief of renal vasoconstriction, the inhibition of excessive secretion of epinephrine and the production of a great vascular bed incapable of vasoconstriction.

Tattooing of the Cornea.

J. N. DUGGAN AND B. P. NANANATI (*The British Journal of Ophthalmology*, July, 1936) describe in some detail their technique in tattooing of corneal opacities. In 25 cases they used gold chloride and tannin, and in four cases gold chloride and hydrazine hydrate. The latter gave a blacker effect. In 14 cases a 2% solution of platinum chloride was employed, fixed with 2% hydrazine hydrate.

Seton Operation in Glaucoma.

AFTER some reference to previous attempts to introduce a seton into the anterior chamber, notably by Zorch and Wood, O. R. Wolfe and M. J. Blaess (*The American Journal of Ophthalmology*, June, 1936) describe their own technique and experience. An eight-millimetre conjunctival incision is made down and nasally five millimetres from the cornea, and the conjunctiva is undermined. A similar flap is made down and temporally. A three millimetre keratome incision is made on one side, and later, when aqueous has reformed, or normal saline solution has been injected to replace the lost aqueous, a similar keratome incision is made on the other side. A straight blunt-tipped lachrymal needle threaded with braided white silk is inserted into the anterior chamber through the keratome incision from the temporal side, and is carefully worked through between the cornea and iris until the tip with the protruding thread emerges from the incision on the nasal side. The silk is grasped with forceps and the needle is withdrawn; each end of the seton is cut off four or five millimetres from the limbus. The conjunctival flaps are closed with a continuous suture without tying. Six cases are described, and the results are considered good. The operation is a pleasing alternative to the enucleation of a blind eye, though not confined to this class.

Local Treatment by Cod Liver Oil.

E. STEVENSON (*The British Journal of Ophthalmology*, July, 1936) advocates the local use of cod liver oil in the treatment of injuries of the eyelids, corneal ulcers and blepharitis. A patient with a severe burn on the inner half of both lids did exceedingly well on this treatment. Notes of over 150 cases convince the author that cod liver oil promotes the growth of natural tissues and inhibits the growth of scar tissue. It should be withheld in the early stages of hypopyon ulcer.

Congenital Head-Nodding and Nystagmus.

R. A. COX (*Archives of Ophthalmology*, June, 1936) gives the differential diagnosis between *spasmus nutans* and congenital head-nodding. In both conditions head-nodding and nystagmus are the most prominent symptoms. In *spasmus nutans* the onset is after birth—at from six weeks to three years of age. In congenital head-nodding the onset is at birth. In *spasmus nutans* recovery invariably takes place, usually before the end of the third year. In congenital head-nodding the condition persists throughout life. In *spasmus nutans* the head movements are slow, inconstant and variable; in the congenital form they compensate for the eye movements. In *spasmus nutans* the nodding precedes the nystagmus and may be monocular; in congenital head-nodding both start together, and the nystagmus is always bilateral. In the congenital form there is an hereditary taint—in the case presented seven of thirty known members of the family were affected. There is no known method of successfully treating the complaint.

Blue Scleras and Hyperparathyreoidism.

A. RADOS AND L. C. ROSENBERG (*Archives of Ophthalmology*, July, 1936) discuss the relation of the syndrome, blue sclerotics, fractures and deafness with hyperparathyreoidism. The normal content of calcium in the blood is between 9 and 11 milligrammes per 100 cubic centimetres. Increase of the blood calcium level with simultaneous increase of the excretion of calcium is called a negative calcium balance, and is characteristic of hyperparathyreoidism. Another feature is a low level of phosphorus in the serum. The skeletal changes in connexion with blue scleras are described as *osteogenesis imperfecta*, a condition involving the connective tissue as a whole, a dysfunction of all matrix-forming cells, including osteoblasts. In hyperparathyreoidism the inability of the bones to store and retain calcium results in decalcification of the skeleton, described by von Recklinghausen as *osteitis fibrosa cystica*. It is generally associated with adenoma of the parathyreoid. There is a lack of uniformity in the laboratory findings in the reported cases of blue sclera. In the two cases described by the authors there were not sufficient variations in the values for calcium, phosphorus and phosphatase to warrant a supposition of endocrine disturbance. *Osteogenesis imperfecta* is hereditary, whereas *osteitis fibrosa cystica* belongs to adult life.

Free Cyst of the Anterior Chamber.

W. H. EVANS (*Archives of Ophthalmology*, May, 1936) reports the history of a woman, thirty-nine years old, who complained of a small dark mass in the left eye for the previous three or four years. Examination revealed a double convex kidney-shaped mass in the anterior chamber, measuring four millimetres by two millimetres. With the patient upright, it rested at the 6 o'clock position; when she moved sufficiently forwards it moved to the 12 o'clock position. After a keratome incision was made the body was successfully removed by irrigation. Only nineteen cases of this condition have been recorded.

Neoarsphenamine in Non-Syphilitic Uveitis.

H. LUCIO (*Archives of Ophthalmology*, May, 1936) advocates the use of intravenous injections of neoarsphenamine in severe cases of iritis and iridocyclitis. It is also useful in sympathetic ophthalmitis. He gives details of ten cases in which favourable results were achieved.

Radiographic Diagnosis of Glioma of the Retina.

R. L. PFEIFFER (*Archives of Ophthalmology*, May, 1936) states that in nearly one-third of the cases in which an eye is removed for glioma an error occurs in diagnosis, the condition being pseudoglioma. Hence any assistance from X ray examination is welcome. Calcareous deposits are common in retinoblastomata, especially in the larger and more

advanced growths. X ray pictures were taken of 20 enucleated eyes, in which glioma was present, and 15 showed calcium deposits. X ray pictures were also taken in 14 cases of probable glioma. In eight cases typical shadows of psammous bodies were revealed, and histological examination subsequently proved the diagnosis of glioma. No calcium shadows were observed in four eyes with pseudoglioma. The shadows of psammous bodies may be distinguished from those of heteroplastic bone in the choriold.

OTO-RHINO-LARYNGOLOGY.

The Effects of Drugs on the Cochlea.

W. P. COVELL (*Archives of Otolaryngology*, June, 1936) has studied the normal mitochondrial content of the cells of the cochleæ of guinea-pigs and the changes produced after drug administration. Quinine bisulphate and sodium salicylate produce mitochondrial changes in the cells of the *stria vascularis* and in the sensory cells of the organ of Corti. More drastic changes in the nature of chromatolysis and a loss of ground substance are evident in the spiral ganglion cells after injections of a solution of sodium salicylate than after injections of a solution of quinine bisulphate. The alterations described usually are more pronounced in the basal turns of the cochlea; but over a longer period of time the apical turns may also become involved. The cochleæ of the fœtal guinea-pig's ears exhibit similar but more profound changes than those of the adult animal.

An Experimental Investigation of Tinnitus.

W. J. MCNALLY, E. A. STUART, T. F. REID AND L. H. MCCONNELL (*The Journal of Laryngology and Otology*, June, 1936) give a preliminary report of their study of nineteen patients suffering from tinnitus. The ages of the patients varied from nineteen to fifty-two years; the duration of the tinnitus varied from a few months to about thirty years; and the diagnosis of ear lesions covered a wide range. Both ephedrine and bellafoline caused improvement in the tinnitus in a greater percentage of cases when given by mouth than when given hypodermically. Since tht hypodermic method of administration was the more accurately controlled, its results were probably more reliable. Stimulation of the sympathetic or depression of the parasympathetic nervous system by drugs, whether carried out separately or simultaneously, had a slightly more beneficial effect upon the tinnitus than did depression of the sympathetic or stimulation of the parasympathetic nervous system. Yet the operation of stellate ganglionectomy (depression or elimination of the sympathetic) caused an improvement in the tinnitus in three out of four cases. All the procedures carried out, with the exception of the sodium-free diet, were directed towards alteration of the cerebral circulation or cerebral pressure. The authors have no direct proof of the nature of the alteration produced, since they made no records of cerebral circulation or pressure during the course of the experiments. It is most likely that the cerebral circulation was affected because the drugs used produced their full physiological effect, and most of these drugs have been shown by other experimenters to have a definite effect upon the cerebral vascular mechanism. In spite of the undoubted alteration of the cerebral circulation, the tinnitus in the majority of the cases in this series was unaffected by the procedures employed.

Bronchoscopy in the Diagnosis and Treatment of Bronchial Carcinoma.

DAVID H. BALLON AND HARRY C. BALLON (*The Journal of Laryngology and Otology*, August, 1936), in a paper dealing with the value of bronchoscopy as an aid in the diagnosis and treatment of bronchial carcinoma, are of the opinion that carcinoma of the bronchus can be diagnosed early on many occasions. Bronchoscopy permits of an early diagnosis more frequently than any other means so far available. It is possible on most occasions to estimate operability by means of the bronchoscope. Too few bronchial tumours have so far been treated early enough by bronchoscopic methods to permit of the evaluation of end results. Published results to date indicate that radium will cause the disappearance of the endoscopically visible portion of the tumour. The oft-reported spread beyond the original site is perhaps only partially attributable to late diagnosis. It is the author's opinion that many bronchial tumours which have thus far responded to bronchoscopic methods of treatment are probably not true carcinomata.

Ætiology and Treatment of Hæmorrhage of the Nose and Throat.

HENRY M. GOODYEAR (*The Journal of the American Medical Association*, August 1, 1936), in a paper dealing with the ætiology and treatment of hæmorrhage of the nose and throat, concludes that reservation must be given in measuring the true value of thromboplastic agents. Ovarian extract is worthy of a trial in hæmophilia; however, blood transfusions are the most reliable. Blood transfusions have no value in *purpura hæmorrhagica*; splenectomy may offer the only relief. Hæmorrhagic telangiectasia responds best to the chromic acid bead. Cocaine, 10%, followed by 50% solution of silver nitrate, is most satisfactory in hæmorrhage from the anterior nasal septum. Gauze packing in the region of the anterior wall of the sphenoid is effective in bleeding from an injured sphenopalatine artery. In intractable nasal hæmorrhages the external carotid artery should be ligated. Insufficient attention is given to adenoid bleeding at the time of the operation. No adenoid operation should be considered complete without retraction of the soft palate and direct examination for bleeding points. Since all branches supplying the tonsils are from the external carotid artery, this would be the vessel ultimately to be tied in an emergency. In suppurative cellulitis following such illnesses as scarlet fever, it is the internal jugular vein and not the carotid vessel which is eroded. Retropharyngeal abscess is relatively infrequent and always a potentially dangerous complication, as is attested by the number of deaths reported. The pus should be aspirated first and then the incision should be made. Immediate pressure over the carotid artery, on the side of the abscess at the time of bleeding, may save the patient during the preparation to ligate the artery. Bleeding from a tonsillar infection with moderate intermittent attacks of bleeding, with no definite pharyngo-maxillary symptoms, justifies the removal of the tonsil and a search for the bleeding point. When sudden severe expulsion of blood occurs in the presence of a retropharyngeal or peritonsillar swelling, either before or after incision, no time should be lost in ligating the common carotid artery on the same side, for at any moment the hæmorrhage may recur, with fatal results. When the pharyngo-maxillary space is distended with pus, it can be drained by incision anterior to the anterior tonsillar pillar or by incision posterior to the posterior tonsillar pillar. Radical removal of the tonsil is justified after incision of a peritonsillar abscess when little or no pus is found, and general distress and neck complications increase rather than diminish. Drainage of a hidden pocket may be thus established. Back circulation may occur through the external carotid after ligation of the common carotid artery. When incision is made for ligation, it is the internal carotid that usually comes into view first, before the external carotid is located. Bifurcation often occurs high, even about the angle of the jaw.

Garlic Breath Odour.

M. A. BLANKENHORN AND C. E. RICHARDS (*The Journal of the American Medical Association*, August 8, 1936) record their experimental work on the fœtid odour that persists for many hours in the breath of one who has eaten garlic or onions. They conclude that onion and garlic breath odours come from the blood by way of the lungs and do not arise "solely from particles of onion or garlic retained in the structures about the mouth". This has been demonstrated by experimental studies on four patients who breathed and ingested food through separate openings. Mouth washes merely mask the offending odour and do not cure it.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Bendigo Base Hospital on August 29, 1936, DR. WALTER SUMMONS, the President, in the chair. Part of this report appeared in the issue of December 19, 1936.

Perthes's Disease.

DR. E. SANDNER showed a boy, ten years of age, who, since he hurt his right leg playing football two years earlier, had had disease of the right hip joint. He had complained of pain in the groin extending to the knee, and it was found that there was limitation of abduction and external rotation; the skiagram suggested that the condition was Perthes's disease. The Mantoux test gave no reaction. Treatment by rest in a plaster spica was commenced; the plaster was removed four and a half months later; another skiagram was taken and the plaster was reapplied and removed again after seven months. Limitation of movements had increased, the thigh muscles were atrophied and adductor spasm was a feature, so the plaster was reapplied for another six months. The child was seen by Dr. D. O. Brown at the Children's Hospital; he was treated for four months in a Balkan frame, and the limb was put up in a Thomas hip splint with slight abduction and improved amazingly. Dr. Sandner demonstrated the series of skiagrams and commented on the degree of involvement shown in them and said that the child was moving about very freely and very comfortably; he invited discussion on the value of immobilizing these patients for such a long time.

DR. C. H. OSBORN commented on the persistence of the spasm of the adductor muscles after prolonged rest and the anomalous radiographic appearances as points which were against the acceptance of the diagnosis of Perthes's disease. Osteochondritis might simulate true coxitis of tuberculous or of coccal ætiology and the diagnosis of Perthes's disease was often confirmed subsequently by the very characteristic slight flattening and very definite fragmentation of the capital epiphysis. In the skiagrams demonstrated there was greater flattening and sclerosis and exaggerated mushrooming of the head. In the course of Perthes's disease over a couple of years he would have expected a dense capital epiphysis and a less mushroomed head with the radiological changes of broadening of the neck and slight rarefaction from disuse. The atypical nature of this case consisted of the prolonged spasm after the long period of immobilization, the excessive rarefaction of the neck and upper portion of the shaft and the grossly mushroomed capital epiphysis. Dr. Osborn said that in his experience of over ten years he had found that Perthes's disease ran a fairly benign course except in the early stages; it soon settled down with immobilization and the characteristic skiagraphic changes appeared. Though Legg himself had indicated that very little treatment was necessary, Dr. Osborn thought that it was rational and logical to avoid weight-bearing during the softening stage by extension and immobilization in a plaster cast; flattening of the head could not be prevented by the plaster cast alone unless there was some extension apparatus attached to the end of it. This was exemplified noticeably in the treatment of tuberculous hip in childhood. After a period of about six months of immobilization and extension a walking Thomas calliper splint could be used for a further six to twelve months to avoid excessive deformity and as a precautionary measure to prevent the osteoarthritis which was liable to occur at about the age of thirty years as a late sequela of Perthes's disease. Dr. Osborn considered that in the boy shown by Dr. Sandner a low grade osteomyelitis might be present, commencing in the metaphyseal region, though a true arthritis did not appear to be present.

DR. THOMAS KING said that he was prepared to accept the diagnosis of Perthes's disease in the case of the boy shown by Dr. Sandner. He agreed with the treatment that had been described by Dr. Osborn for the typical condition. He considered that a lateral skiagram should have been taken to supplement the information obtained in the antero-posterior picture. He did not favour treatment in a plaster cast because of the muscle wasting and exclusion of sunlight which it involved. It was noteworthy that the head of the femur was very susceptible to injury and that the causes of most of the diseases that affected it were unknown. Dr. King referred to the condition of fragmentation of the neck of the femur, which had been described recently; the appearances closely resembled those of the more usual osteochondritis of the head of the bone. In traumatic dislocation of the head tremendous osteoarthritis might follow; again, radiographic changes might be apparent on both sides and the patient's complaint might be limited to pain on one side only. Dr. King gave an interesting description of the subtrochanteric osteotomy of Lorenz, in which by means of Stille's special Gigli saw the shaft could readily be divided without the shock of hammering and without splintering; the shaft was displaced medially under the lower portion of the head of the bone from which it was separated by the capsule and through which it took the weight, and the patients could soon walk without pain. He preferred not to put the leg in the usual plaster spica, but rested it snugly on a firm half mattress made of "Latex" rubber. Under anæsthesia it might be necessary to slip the divided shaft back into position at the end of the first week and again at the end of the second week, after which it usually stayed in position. Dr. King also described the Schanz osteotomy, in which the bone had to be separated opposite the lesser trochanter and fixed by means of a pin into the lateral wall of the pelvis below the acetabulum with the leg in adduction.

Advances in Pre-Operative and Post-Operative Treatment in the Past Decade.

DR. D. W. NEVILLE introduced a discussion on the progress of a decade in pre-operative and post-operative treatment. He said that, recognizing that every change was not an advance, he had selected certain alterations that had proved to be advances. To evaluate progress he wished first to reflect on the directions in which ten years earlier medical practitioners looked for improvement and then to consider what progress had been made in these directions. The discomforts and the dangers of operations were the main directions in which progress was desired. The pre-operative discomforts were fear, starvation and purgation, and the post-operative discomforts were pain, persistent vomiting and abdominal distension. The main dangers were shock, ileus and uræmia.

Dr. Neville then referred to the measures which had been adopted for the prevention of serious developments. Patients had been starved for a considerable time, perhaps overnight and for the whole of the day, until the operation commenced in the afternoon; they had been purged on the previous day with loss of sleep during the night and came to the theatre almost in an exhausted condition; the sleepless night was in part due to fear of the outcome of the operation and in part to the purgation; all the conditions were present to favour the production of post-operative shock. Patients were no longer starved; they were given glucose or barley sugar for several days before the operation to increase the liver store of glycogen, and they were not purged continuously, though they might have an enema on the night before the operation. So far as the fear of anæsthesia was concerned, a great advance had been made by the widespread use of basal anæsthetics. Ten years earlier it was customary to give premedication with morphine and atropine; "Avertin" had a brief popularity and was not yet quite a thing of the past, but it had a small margin of safety as a medium for putting people to sleep and he had heard of several goitre patients who did not wake up. Paraldehyde was safer and was very effective; six to eight drachms were administered rectally often in conjunction with morphine and hyoscine; very little inhalation anæsthesia was required and commonly patients did not wake up for eight or nine hours after the operation; they had very little pain or vomiting, with resultant reduction of the discomforts of operation.

More recently "Nembutal" had been advocated, though Dr. Neville had had very little experience of it; a capsule containing one and one-half grains given the night before operation removed pre-operative fear and allowed the patient to have a good restful sleep. One hour before the operation one or two capsules of "Nembutal" were given and the effect was not to put the patient to sleep, but a considerable reduction in the amount of ether was possible, patients were easily kept in a state of surgical anæsthesia, and post-operative vomiting was almost negligible; besides, it was easier to give than paraldehyde by the rectum.

Passing from these measures of overcoming discomforts, Dr. Neville next considered the treatment when shock and ileus were developing, and intimated that he did not propose to discuss uræmia. Shock was due mainly to cold and to pain and to loss of blood or of any other body fluid. Loss of body heat in the operating theatre was caused by considerable exposure for a considerable length of time; the operating theatres were kept warm and the patients were kept covered, so that this loss was greatly reduced. Overheating of the operating theatre had not proved successful, and even ten years previously the use of such conditions was being discontinued. There had always been unanimity that the administration of fluids combated shock; ten years earlier rectal administration of saline solution was used, and in addition subpectoral or submammary injections of saline solution were frequently given; it had been quite a common sight to see two or three patients having these injections at the same time in the same ward. It was now considered that glucose with saline solution given by the rectum and also subcutaneously was an improvement. In 1927 Levi had published reports of the taking of blood-sugar curves which showed the post-operative rise and that the greater the shock, the longer it took for the blood-sugar to return to the normal. There was actually an indication that the tissues were unable to utilize glucose during shock. It was an advance to give insulin in a dose of one unit for every two or three grammes of glucose. With the insulin, 5% to 10% glucose in normal saline solution was given intravenously. In the presence of severe shock threatening life it was found that the systolic blood pressure fell below 100 millimetres of mercury, and it was recognized that when the systolic pressure fell to 90 millimetres it was on the verge of an extremely rapid fall presaging death. In such circumstances the administration of one pint of hypertonic saline solution (6% to 10%), given intravenously, usually proved a rapid restorative to the volume of the pulse and the blood pressure and tided the patient over the period of shock.

Dr. Neville then referred to ileus as another bugbear of operation to which a considerable amount of thought had been given. He stated that in every case of abdominal distension within two weeks of operation it was necessary to make sure whether some mechanical obstruction had occurred. In those cases in which it was obvious that the distension was not due to mechanical obstruction, the circumstances might be that the operation was performed for advanced appendicitis with the presence of a little fluid in the peritoneum and closure of the abdomen without drainage; the patient might have abdominal distension and be unable to pass flatus without actually vomiting. Such a state of affairs might be compared with the case of appendicitis, in which the doctor was not consulted until the fourth day, when he might decide not to operate though he could feel a mass and knew that diffuse peritonitis was present. Dr. Neville considered that such a patient had just as good a chance of recovery without operation. The treatment would be to nurse the patient in the half-sitting position and not to allow him to have any food and no drugs or purgatives, but to give him plenty of water. This treatment could be continued while the patient was not vomiting and the pulse was not going up; and a good percentage of patients treated along these lines did quite well. No bowel action might occur for several days and yet the condition subsided and all was well. When the appendix was removed in such a case, the condition of the peritoneum was just the same and similar treatment was suitable; the patient should not be tormented with purgatives or given repeated high enema and should not be fed; he should be given water freely, in the form of glucose solution or sweetened barley water, until the abdomen was slack and he started to pass flatus. In the other type of ileus with vomiting after the appendix had been removed paralytic ileus had to be considered. There had been a change of opinion during the past ten years concerning the management of this condition. Eight or ten years previously divided doses of calomel, for instance, one grain every hour for four hours, would be given on the first day as soon as the condition was suspected, followed by turpentine enemata or ox-bile enemata to compel the bowel to function. The results were bad; the patients became emaciated as the result of persistent vomiting and usually died. Spinal anæsthesia was a notable advantage. The effect was to relax the sphincters throughout the alimentary tract and to allow the resumption of peristalsis. The administration of acetyl choline enabled the extended vagus nerve endings to transmit stimuli to the bowel musculature. Operation such as jejunostomy was performed in the belief that if distension of the bowel was relieved, peristalsis might resume; but all these operations left much to be desired.

A lot had been learned about these conditions in the past ten years. In cases of definite obstruction, such as strangulated hernia, it was wise to avoid any possible delay; the amount of vomiting was misleading and pain might not be severe. Abdominal distension of the step-ladder variety appeared only when considerable damage had occurred; it was not merely a sign of obstruction. Obstruction should be diagnosed, or at all events operation should be undertaken, before the abdomen became distended. Another fact that was recognized was that rapid emptying must be avoided in the face of distension because it immediately caused shock. Vigorous post-operative stimulation treatment had largely been abandoned because it did not offer anything effective.

Dr. Neville summarized the procedure that had emerged from the discussion and experiences of the decade, by stating that in the post-operative treatment no purgatives were given and if distension occurred one high turpentine enema might be given, as it might nip ileus in the bud. It often failed to do so, however, and at all events it was generally accepted that a period of rest, say twelve hours, should be allowed, and that in this interval there were two measures of hope that could be applied. One was the administration of one pint of 6% saline solution by the intravenous route to make good the loss of fluid and the loss of chloride and to provoke vigorous peristalsis which would often make a second enema unnecessary. The other procedure was to pass a small stomach tube which might be left in for several hours; so long as mucus and bile only could be recovered through the stomach tube and the pulse rate was not rising he felt satisfied to wait. The saline solution was sometimes given by the continuous drip method, and it was now recognized that the temperature of the saline solution did not matter very much. If at the end of twelve hours there had not been a bowel action, a second high turpentine enema would be given. If it failed to produce a bowel action, a high jejunostomy should be undertaken and a small rubber catheter should be stitched into the bowel. Dr. Neville commented on the increasing tendency to use continuous saline injections and predicted that in future they might see as a regular thing a needle fixed into the arm at the commencement of a serious operation. It would be used for the basal anæsthetic, and throughout the operation saline solution would be introduced by the continuous drip method. In this way the difficulties might be overcome by diminishing the occurrence of ileus and shock.

DR. A. E. COATES, in congratulating Dr. Neville on the interesting way in which he had opened the discussion, noted that he had practically confined his remarks to the abdomen, as it was impossible to cover the whole ground. Dr. Coates said that enthusiasm was waning at the Royal Melbourne Hospital so far as the use of basal narcosis was concerned; they had gone back to the use of morphine and atropine or chloretone and atropine; he used morphine and atropine as a routine in head cases. As a basal narcotic paraldehyde was used quite a lot, but Dr. Coates often

doubted whether it was as safe as people said it was; he remembered the case of a woman with a cerebellar tumour who had expelled the paraldehyde from the rectum into the bed, and, in the belief that none had been retained, more was given, and the patient came to the operation table dead. Dr. Coates said that the objection to anything put in *per rectum* or intravenously was that the effect could not be cut off; if any damage was done it could not be shut off and tragedies occurred.

Dr. Coates joined issue with Dr. Neville concerning the causation of shock; it was due to slow leakage of blood. He was in the habit of keeping records of blood pressure and of the pulse during the operation, and this fact came out clearly. He paid a great deal of attention to very careful hæmostasis in each stage of an operation. In jejunostomy for obstruction Dr. Coates was never optimistic about the amount of fluid that would come away; he would be very happy to get as much as one pint of fluid in twenty-four hours. He found that it was a very difficult problem to decide how to recognize an ileus in the early stages, or how to recognize that he was dealing with a case of mechanical obstruction. A piece of ileum might be picked up and caught in the purse-string suture, and the lower part of the ileum might become glued down and go on to obstruction. The patient would have a colicky pain as gas gurgled through the ileo-colic valve. When the small valve distended, it straightened out, and at the corners it kinked acutely, and, superimposed on slight obstruction, they had a series of obstructions. The original piece of bowel might become unstuck by the pressure of the distension and yet at the autopsy the finding would be death from paralytic ileus. Dr. Coates recommended the application of radiant heat to the abdominal wall and the passage of a rectal tube; if the patient complained of a salty taste in the mouth dilute hydrochloric acid should be given in a mixture.

DR. C. H. OSBORN said that he thought that the subject of basal anæsthesia had been passed over rather lightly; there had been a tremendous advance in surgery in the past ten years as the result of the judicious use of the newer forms of anæsthesia. Dr. Osborn believed that perhaps within the next ten years a great future lay in the production of anæsthesia which was perfectly safe, and, commencing five to ten hours before operation, would persist for seven to ten days afterwards. This idea might sound fatuous, but it was still a reasonable demand, and he felt sure that it was going to come. The use of basal anæsthetics and local anæsthesia had proved that post-operative shock could be minimized, and with the passage of time he for one used them more and more frequently, and, in the public hospital with which he was associated, there was very general use of local and spinal anæsthetics in major surgery and especially in abdominal surgery. It gave him a great deal of comfort to see patients reading the paper the day after so serious an operation as subtotal gastrectomy had been performed on them. The purpose of his remarks was to stimulate those who did not use basal anæsthetics and local anæsthesia to try them; he conceded that the surgeon must be prepared to handle the tissues more gently and to take longer over the operation.

Dr. Osborn, in referring to the tremendous vogue for intravenous therapy, expressed the opinion that this definitely could be overdone; in particular, he referred to continuous administration of saline solution and to the administration of blood or of glucose solutions, which, he said, were not necessary in a large number of cases in which they were used, and sometimes might be followed by disastrous results. He emphasized the ease with which fluids could be introduced by mouth and absorbed readily. He advocated the administration of water by mouth in the operating theatre and of four ounces of Hurst's Number 1 diet right away on the return of the patient to the ward; if a patient was fed soon after operation he would not get distension.

DR. L. HARTNETT said that he still used paraldehyde, but did not think that it was as valuable as morphine and hyoscine with local anæsthesia because the patients were liable to become restless. Often there was no vomiting at all after operation, though at times the patients might vomit once; almost invariably several hours after operation they asked when they were going to have the operation.

DR. E. SANDNER expressed the opinion that local anæsthesia should be used more frequently at Bendigo. He had found that "Avertin" was a satisfactory basal anæsthetic, as was "Nembutal" given intravenously, but he did not think that paraldehyde was as good. Though he had heard that hyoscine at times induced delirium, he had not yet encountered an example.

"Funny Turns."

DR. S. O. COWEN read a paper entitled: "Funny Turns" (see page 58).

DR. H. F. MAUDSLEY thanked Dr. Cowen for his extremely interesting paper and said that he would limit his contribution to the discussion to the psychiatric cases he encountered in practice in which the relatives referred to certain incidents as "funny turns"; very often he found that these incidents were not so mild as those that Dr. Cowen talked about. An early adolescent psychosis was often ushered in by a turn in which the subject sat in a corner and muttered or might hear voices or have other hallucinatory experiences; these would turn out to be something serious later on. One often saw or heard of such occurrences in pre-psychotic subjects for weeks or months before serious symptoms supervened, for example the symptoms of early *dementia præcox*. A patient with uncinate gyrus tumour would have these curious hallucinations of smell or the *déjà-vu* turns that had been described by Kinnear Wilson. Curious effusions occurred in the cerebellar regions, probably owing to inflammatory changes, which produced strange effects at times. He had been interested to hear of claims that cervical sympathectomy had cured migraine, and would like to know of any local experiences.

DR. WALTER SUMMONS, in concluding the meeting, expressed his gratitude and the appreciation of the visitors to members of the Bendigo Subdivision. He referred to the wife of a doctor who was free of funny turns only when she was going for a sea voyage; and to another woman who was frequently prostrated and was relieved only by injections of morphine; he managed to keep her free of turns by limiting the injections to one every three months.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Gay, Keith Roy, M.B., B.S., 1936 (Univ. Sydney), 494, Mowbray Road, Lane Cove.

Medical Societies.

THE MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, Melbourne, on August 12, 1936, DR. C. H. OSBORN, the President, in the chair.

The Treatment of Bronchiectasis and Pulmonary Abscess.

Following the presentation of several patients with bronchiectasis and one who had recently been subjected to lobectomy, the meeting took the form of a symposium on the subject of the surgical treatment of bronchiectasis and pulmonary abscess.

DR. L. P. WAIT showed a boy, aged thirteen years and nine months, who had attended the Children's Hospital since March, 1930. He had a history of constant cough from the age of two years. He had not had pneumonia,

whooping cough or measles when he first attended, but examination revealed the presence of a patch of amphoric breathing with râles at the base of the left lung, and there was slight clubbing of the fingers. At that time it was reported on radiological examination that fibrosis was present with bronchiectasis at the base of the left lung; the appearances at the base of the right lung were within normal limits, and no evidence of pathological change in the antra was found. The blood serum did not react to the Wassermann test. Some time later skiagraphic evidence was obtained of bilateral sinusitis, and by antral lavage thick pus was obtained from both antra. Dr. Wait said that the patient had attended the out-patient department regularly every month until November, 1935; the complaint was always of cough; he apparently had an attack of acute bronchitis in 1931, in which signs were noted at the bases of both lungs, but the signs at the base of the left lung, râles, rhonchi and tubular breathing had been constant throughout the whole period of observation. In 1933 the sinuses were drained, as the patient was considered to be too young to undergo the radical operation on his antra. In a skiagram taken in 1934 a honeycombed appearance was seen at the base of the left lung, and a year later a lipiodol investigation resulted in the diagnosis of a very definite bronchiectasis of the saccular type in the lower lobe of the left lung.

DR. W. W. MCLAREN showed a lad, seventeen years of age, who had first attended at the hospital in 1926. Though there was no antecedent history of measles or of antral disease, the child had had whooping cough, scarlet fever, nephritis and otitis, and his tonsils and adenoids had been removed. When seen again in 1928, he still suffered from chronic otitis, a chest deformity was noted, and cough and night sweats had developed. He was admitted to hospital for several months and had a large pleural effusion at the right base, from which a lot of straw-coloured fluid was withdrawn, and a small pulmonary cavity, from which some thick pus had been obtained. The radiological reports mentioned the presence of calcified glands in each hilum, the right basal effusion and, at a later stage, fibrosis. In 1929 fibrosis and cavitation of the right base were noted in the skiagrams, and on the introduction of lipiodol the saccular cavities at the right base were outlined. The radiological appearances of slight mucosal thickening of the antra were recorded at the same time. In 1934 the boy had gross saccular bronchiectasis of the lower lobe of the right lung and fibrosis at the left base, and he was transferred to the Royal Melbourne Hospital. The most recent examination was made on August 6, 1936, when there was radiographic evidence of gross bronchiectasis at the right base, but no advance of the disease on the left side. By means of a bronchoscope a good deal of pus was found in the left bronchus and pus actually welled up in the right bronchus. The lad was well nourished and weighed 53·5 kilograms (eight and a half stone). Some curving of his finger-nails was noted and his breath was foul-smelling; he usually coughed up a cupful of pus each day, and the signs at the right base were definite.

DR. MOSTYN L. POWELL showed a boy, seven years of age, whose illness had started four and a half years earlier with an attack of whooping cough, followed by bronchitis and then scarlet fever. Three months later he had bronchopneumonia, and this condition had recurred every three months. Since an attack of measles and bronchopneumonia early in 1935 he had had a persistent cough with constant production of sputum. Radiological investigation on June 4, 1935, established the diagnosis of left basal bronchiectasis with collapse of the lower lobe and displacement of the mediastinum and gross involvement of both antra. Bilateral radical antrostomy was done soon afterwards and treatment by postural drainage was commenced. The patient's condition seemed definitely to improve, but he still had a good deal of cough and sputum.

Dr. Powell said that certain points were outstanding since treatment had been commenced. The mother was pleased with the lad's improvement and said that he ate well and played in a normal manner; he still had a good deal of morning cough, and with postural drainage about half a cupful of thick yellow sputum resulted. This amount of sputum was not becoming less, and in April, 1936, he had another bout of bronchopneumonia; the left lower lobe was still collapsed and the mediastinum displaced; the right base was still free of bronchiectasis. Dr. Powell invited a discussion of the prognosis and treatment either by lobectomy or by more conservative measures.

DR. E. S. J. KING discussed, in introduction, the essential morphological changes in bronchiectasis with illustrative lantern slides, showing the improbability of any material cure and the necessity for definite treatment.

The treatment was divided into two main groups: (i) drainage, (ii) removal of the diseased part. Drainage in its various forms, postural, bronchoscopy and by cautery lobectomy were discussed and their inadequacy was emphasized.

Dr. King said that removal of the diseased area, the operation of lobectomy, might be performed by two methods: (*a*) in two or more stages or (*b*) in one stage. The various forms of the two-stage procedure were described briefly, and finally one-stage lobectomy was illustrated. It was emphasized that this operation was still being modified, and Dr. King mentioned the problems of incisions in the chest wall and methods of dealing with the hilar stump.

Finally, some of Dr. King's experiences with thoracotomy for various conditions were described, and it was pointed out that, if allowance was made for the physiological characteristics peculiar to the chest, its rigid cage and the negative pressure, thoracotomy was a relatively safe procedure. Two essential precautions were preliminary pneumothorax and post-operative negative pressure drainage. If these were recognized, then thoracotomy was little, if at all, more dangerous than laparotomy.

DR. IAN J. WOOD showed a boy, fourteen years and six months of age, who had developed a cough two weeks after tonsillectomy eighteen months earlier; the cough had persisted till the operation sixteen months later; the cough had been accompanied by offensive sputum, repeated attacks of pyrexia, pallor and general malaise, with a failure to gain in weight. Three months later, physical signs of fibrosis and cavitation at the right base were confirmed by a lipiodol examination carried out by Dr. Reginald Crisp and by a bronchoscopic examination by Dr. Raymond Hennessy. Two months later, after a month of postural treatment, lobectomy was performed; this was followed by an empyema which was drained, and two months after the operation there was only a slight discharge from the wound; there was no cough or sputum, the breath was sweet, the colour was good, and the patient had gained 4·5 kilograms (ten pounds) in weight.

DR. W. E. A. HUGHES-JONES explained the surgical procedure adopted in the case of Dr. Ian Wood's patient—a formal one-stage lobectomy made tedious by dense adhesions; Dr. Howden had administered nitrous oxide anæsthesia. A good approach was obtained by the incision in the fifth interspace. The lobe was found to be fleshy and its edges ruptured easily in the fingers and discharged foul pus; the lung tissue in the pedicle was so friable that the oversewing was inadequate. Dr. Hughes-Jones said that, as was to be expected, pleural sepsis became evident in the second week, but the closed negative pressure drainage limited its extent.

DR. REGINALD WEBSTER, who had examined the removed lobe and reported that it contained one main cavity and several subsidiary cavities, which were encased in dense fibrous tissue, demonstrated the specimen at the meeting.

DR. C. J. O. BROWN said that the cases that had been presented were interesting and instructive, and he would like to offer his congratulations to Dr. Ian Wood and to Dr. Hughes-Jones for the way in which the case had been handled and the successful outcome of the lobectomy. Dr. Brown referred in some detail to the patient he had treated by lobectomy almost two years earlier. He had reported this case in *The Australian and New Zealand Journal of Surgery* dated October, 1935. He realized now that operation had been delayed too long in this case, because, before operation, evidence had been obtained of early tubular bronchiectasis at the right base, as well as cavitation at the left base. After the operation the condition at the right base had advanced considerably, which was dis-

appointing. The mortality risk of the operation of lobectomy was approximately 14%, but no doubt, with improved selection of cases and earlier operation, this mortality would be considerably reduced. Bronchiectasis was a disease of young people and the majority died before the age of forty years if treated conservatively; it was still too soon to know with certainty what degree of improvement there would be after lobectomy, but the hopeful attitude which had been adopted at such a centre as the Brompton Hospital in England was quite justified by the progress of the patients. If conservative treatment was adopted from an early stage, the ultimate prognosis was just as bad as it was for the patients seen at the later stages. Dr. Brown was sure that in properly selected cases lobectomy should be done early while the condition was limited to one lobe. He drew attention to the absence of reports in the literature of post-operative lipiodol examinations, and inferred that valuable prognostic information could be obtained by such examinations. With reference to the patient who had been shown by Dr. Powell, Dr. Brown thought that the right lobe should be aspirated, as the moist sounds on this side might be due to a spill-over from the bad left side, the lower lobe of which was collapsed. After aspiration, if the lipiodol introduced produced a picture of a good bronchial tree, it was reasonable to assume that the moist sounds were due to a spill-over. The most favourable cases for operation were those following ingestion of a foreign body followed by bronchostenosis and true unilateral bronchiectasis.

DR. CLIVE FITTS said that in the type of saccular bronchiectasis which had been described by Dr. King there was only one possible cure, and that was operative removal. Dr. Fitts had been at Brompton at the time when current thought in this matter was comparable with that existing in Melbourne at present; the physicians were disinclined to refer patients for operation. In that year (1932) he doubted whether as many as twenty of these operations were done in England, but with the exception of two or three they were done at Brompton. The indications for the operation were that the conditions should be defined as unilobar after radiological investigation, that the patient should have a chronic cough with production of an excessive amount of sputum and perhaps a history of hæmoptysis, the presence of continued elevation of temperature and the evidence of parenchymatous disease of the lung. The number of cases for operation should be limited to start with. It would be regrettable if anything like all the patients with bronchiectasis were submitted to operation. Dr. Fitts referred to the after-effects of lobectomy and stated that a blind sac was left. He mentioned lipiodol investigations of one case two years after operation; a blind sac was present and the patient was free from symptoms. He thought that the patient upon whom Dr. Hughes-Jones had operated was a very inspiring and important case; great care had been taken over the pre-operative treatment and the after-treatment. It was absolutely necessary to get expansion of the lung after operation; failure to get expansion led to further bronchiectasis. He mentioned the introduction of a needle to draw off some air over the upper lobe to help the lung to expand. Dr. Fitts considered that one surgeon should get special facilities at each hospital to develop the necessary technique and to follow up the patients and study the natural history and physics of the chest.

DR. J. G. WHITAKER said that he had been privileged to see Dr. Hughes-Jones operate on the patient that had been presented, and he thought that modesty had prevented Dr. Hughes-Jones from doing himself justice in failing to describe adequately the difficulties that he had encountered and overcome. If that patient could get better, Dr. Whitaker thought that there was hope for any other patient who might be operated on. The ligation of the pulmonary pedicle and the oversewing of the bronchi and vessels had presented a very awkward problem. Dr. Whitaker wondered whether it would be possible to ligate them *en masse* and leave it. He had also realized the importance of the drainage after operation, and had been very impressed with the measures that had been adopted, and he offered his congratulations to Dr. Hughes-Jones and Dr. Ian Wood. Dr. Whitaker emphasized the cooperation that was needed between the surgeons and the physicians to secure such results, and he felt convinced that in such cases tinkering around by medical means was just nonsense.

DR. W. W. MCLAREN and DR. H. BOYD GRAHAM indicated that they would watch for suitable cases in the out-patient department for treatment by lobectomy, and stated that by conservative measures unsatisfactory results were being obtained.

DR. DOUGLAS GALBRAITH stressed the importance of more careful and prolonged convalescence of young patients suffering from respiratory infections; by such measures a great number of cases of severe bronchiectasis might be prevented.

DR. D. O. BROWN said that there should be more lipiodol investigations and that these should be carried out by a skilled bronchoscopist who would suck the exudate out of the bronchial tree before the introduction of the lipiodol. Dr. Brown referred to the severity of interstitial pneumonia, which often proved fatal under the age of two years; in such cases dull patches could be detected, but no frank empyema found; at autopsy the whole lobe of a lung or more would be found to be mushed up. He regarded such a condition as this acute suppurative bronchiolitis as the precursor of a great number of the cases of bronchiectasis. With reference to the selection of cases for operation, there was no room for the contention that an undue number of operations would be attempted; the surgeons would not be prepared to do a lobectomy on any other than a carefully selected patient. Dr. Brown considered that he had seen only one suitable patient in the past two years, and that was the patient shown by Dr. Powell that night, whom he regarded as eminently suitable for lobectomy. Probably fewer than one in twenty of the available patients with bronchiectasis would be operated on.

DR. C. H. OSBORN, after expressing his gratification at the success of the discussion, and before calling upon Dr. Hughes-Jones to reply to questions, referred to a small practical point that had been helpful to him in the surgery of empyema. He had found that, when trying to get negative pressure drainage, the tube was liable to become loose, allowing air to get in. He had used with success a simple device made from a piece of the inner tube of an ordinary motor tire. The modern tube was moulded and when cut could be fitted accurately to the chest wall; a hole was punched one-eighth of an inch in size smaller than the calibre of the piece of tubing to be placed in the wound. The drain tube was forced through this hole and the motor tube was pasted onto the chest wall with mastic gum applied to the inner rough surface. This device had enabled him to maintain negative pressure drainage for as long as a week or ten days without reapplication, and he thought that something of the sort might be useful in the after-treatment of lobectomy.

Dr. Hughes-Jones, in reply, said that the trouble he had experienced with an anterior effusion in the case of the boy he had presented had cropped up in various reported cases. The suggestion that Dr. Fitts had made about drawing off air after the operation was a good one, but it had been done by several surgeons without completely solving the problem. The posterior tube was liable to get sealed off, and it might be a good idea to put in an anterior tube at the time of the operation in preparation for the effusion. Dr. Hughes-Jones advised Dr. Whitaker that he thought it was better to avoid mass ligation if possible.

Correspondence.

REFLECTIONS UPON CHRONIC AURAL SUPPURATION.

SIR: A short time ago I listened to a very instructive discussion upon the surgical treatment of chronic aural suppuration and from that exchange of opinions a few thoughts emerge.

Firstly, there is very general agreement among present-day otologists that surgical interference in chronic aural suppuration aims at placing the patient in as safe a position as possible from all future complications which may endanger life, and by the removal of all diseased structures, leaving intact the essential conducting apparatus of hearing, to obtain, if possible, a perfectly dry and functioning ear. Several variations of a modified mastoidectomy are employed by different surgeons in the endeavour to effect these objects. So far there is agreement now among practically all surgeons.

Should, however, the modified operation, in spite of careful performance and after-treatment, fail to achieve a perfect technical result, inasmuch as some discharge, perhaps very slight indeed, remains permanently, then some surgeons seem prepared to advise (if the patient will consent) that the modified operation should be transformed into a radical one, with, of course, for the patient permanent and for all practical purposes complete deafness in that ear, and unfortunately not even then any guarantee that all discharge will cease.

Such an attitude as a general principle cannot, in my opinion, be reasonably maintained and is surgically unsound. The mechanism essential for air conduction is far too precious and valuable to be destroyed, even in the endeavour to obtain a perfectly dry ear, and even in those cases where the other ear happens to be quite sound.

The modified operation may fail to effect a complete cessation of discharge, but the patient at any rate is made reasonably safe, and what is supremely important, possesses an organ which functions to a greater or less degree. Far better a technically imperfect operation with hearing than the most perfect result and practically complete deafness. In the former case the patient has every opportunity of being a useful member of the community, economically and socially, and of enjoying personal pleasure and happiness; in the latter he is sorely handicapped and made miserable by his defect. In short, the radical mastoid should be an extremely rare operation, whether performed after a modified mastoidectomy or as a primary operation, and should be reserved for very special and unusual conditions only.

The second, and to me by far the more important, thought is that every possible means and all knowledge at present available to us should be used in their prevention. Few fields of endeavour would yield such a rich harvest. Adenoids and tonsils are, of course, well attended to and all reasonable precautions, by isolation and treatment, are taken with the more severe exanthemata; but what of the common cold, the cause of so much ill-health and of nasal sinus and middle ear infections?

The medical profession can claim little knowledge of, and certainly has no reasonably efficient treatment for, this scourge of the human race. Nose and ear specialists might well occupy some of their time to much worse purpose than in collating their present knowledge upon the subject and in laying down simple rules for treatment, which everybody could begin to use immediately the first symptoms of nose or throat catarrh appeared, and thus the danger of infecting others could be lessened.

Have we no wealthy citizens and no governments willing to provide funds for research work upon the common cold and especially to endeavour to find an efficient treatment for it?

All governments would be richly repaid in spending large sums annually upon building up the health of the people and especially of the children against infections of all kinds by a daily adequate supply of all the necessary vitamins.

If all our food were less refined and more scientifically cooked and therefore allowed to possess most of its vitamin content, or if we all took a small amount of vitamins daily with our present diet, we would go a long way towards increasing our resistance to infections of all kinds, and particularly to colds and the often accompanying nasal sinus and aural infections, and we might even obtain some explanation of the frequency of appendicitis during the last thirty-five years, and perhaps of many other problems which now puzzle us.

All our energies devoted to the prevention of these blighting aural and nasal infections would confer vastly greater blessings upon humanity than the most perfect curative operations, however successfully conceived and executed.

Yours, etc.,

ERNEST R. SAWREY.

31, Collins Street,
Melbourne, C.1,
December 7, 1936.

MALARIA.

SIR: The late Sir Walter Fletcher, of the Medical Research Council of Great Britain, estimated the number of persons infected with malaria as roughly 650 millions, or one-third of the population of the globe.

The relevant data were sent to me in a letter dated November 23, 1932. Where malaria is endemic the death rate is 3‰ to 4‰, and as the death rate was then estimated at two millions (now three and a half millions), his estimate was conservative.

The International Health Board of the Rockefeller Foundation express the opinion, based on reports, that the incidence in tropical regions is increasing. In Nigeria the parasite rates for adults and children are higher than they were thirty years ago. The ascertained parasite rates for infants under one year is over 90%, in spite of a large expenditure on anti-malarial measures.

Colonel James, F.R.S., allows 200 grains of quinine as a fair average for the treatment of one case, and estimates that 47 million cases are treated annually, of which seven and a quarter million are in the British Empire and five and a half million in British India. The annual expenditure in quinine in the world is £2,500,000, in the British Empire £370,000, and in British India £288,000. On the whole, the mosquito is more than holding his own in this conflict.

Ceylon has a population of five millions, of whom 200,000 died recently from malaria, but formerly the population was seventeen millions, but malaria eliminated twelve millions.

Greek civilization was probably submerged from this cause, and Mark Antony, Nebuchadnezzar, Cœur de Lion and others lost armies from this source. Field Marshal Lord Allenby was the only commander who realized the risk in Syria and Palestine, and made his dispositions accordingly.

If malaria gets a grip of Australia people will be sorry for themselves. For reasons set out in other communications I think it unlikely, but not impossible; but every care should be taken when a case makes its appearance.

Yours, etc.,

JAMES W. BARRETT.

103-105, Collins Street,
Melbourne, C.1,
December 29, 1936.

Obituary.

GRAFTON ELLIOT SMITH.

WE regret to announce the death of Sir Grafton Elliot Smith, which occurred on January 1, 1937, in London.

Books Received.

THE LAST THIRTY YEARS IN PUBLIC HEALTH: RECOLLECTIONS AND REFLECTIONS ON MY OFFICIAL AND POST-OFFICIAL LIFE, by A. Newsholme, K.C.B., M.D., F.R.C.P.; 1936. London: George Allen and Unwin Limited. Demy 8vo, pp. 410, with illustrations. Price: 15s. net.

APPLIED DIETETICS FOR ADULTS AND CHILDREN IN HEALTH AND DISEASE, by S. Blum, A.B., M.S., M.D.; 1936. Philadelphia: F. A. Davis Company. Royal 8vo, pp. 419.

INDIVIDUAL PSYCHOLOGY MEDICAL PAMPHLETS; Number 16: The Place of Psychology in the Medical Curriculum and other Papers, by W. Langdon-Brown *et alii*; 1936. London: The C. W. Daniel Company Limited. Demy 8vo, pp. 63. Price: 2s. 6d. net.

MODERN TREATMENT AND FORMULARY, by E. A. Mullen, P.D., M.D., F.A.C.S., with foreword by H. C. Wood, junior; 1936. Philadelphia: F. A. Davis Company. Demy 8vo, pp. 707.

PRACTICAL PHYSIOLOGICAL CHEMISTRY FOR MEDICAL STUDENTS, by G. M. Wishart, D. P. Cuthbertson and J. W. Chambers; 1936. Glasgow: John Smith and Son (Glasgow) Limited. Crown 8vo, pp. 125. Price: 3s. 6d. net.

NOTES ON CLINICAL LABORATORY METHODS (Standing Committee on Laboratory Methods, University of Glasgow); Third Edition; 1936. Glasgow: John Smith and Son (Glasgow) Limited. Crown 8vo, pp. 86, with illustrations.

LEAGUE OF NATIONS: QUARTERLY BULLETIN OF THE HEALTH ORGANIZATION, Volume V, Number 3, September, 1936; 1936. Geneva: Publications Department of the League of Nations. Royal 8vo.

SCALPEL AND SWORD, by Sir James Elliott, M.D.; 1936. Australia: Angus and Robertson Limited. Demy 8vo, pp. 215. Price: 7s. 6d. net.

TREATMENT IN GENERAL PRACTICE: THE MANAGEMENT OF SOME MAJOR MEDICAL DISORDERS (Articles Republished from The British Medical Journal); Volume II; 1936. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 440, with four plates. Price: 10s. 6d. net.

ON THE INCIDENCE OF ANÆSTHETIC COMPLICATIONS AND THEIR RELATION TO BASAL NARCOSIS, by C. J. M. Dawkins, M.A., M.D., B.Chir., D.A., with foreword by J. Blomfield, O.B.E., M.D.; 1936. London: John Murray (for the Middlesex Hospital Press). Demy 8vo, pp. 56. Price: 5s. 3d. net.

RECENT ADVANCES IN ALLERGY (ASTHMA, HAY-FEVER, ECZEMA, MIGRAINE, ETC.), by G. W. Bray, M.B., Ch.M., M.R.C.P., with foreword by A. F. Hurst, M.A., M.D., F.R.C.P.; Third Edition; 1937. London: J. and A. Churchill Limited. Demy 8vo, pp. 532, with illustrations. Price: 15s. net.

DON'T BELIEVE IT! SAYS THE DOCTOR, by A. A. Thomen, M.D.; 1936. Australia: Angus and Robertson Limited. Demy 8vo, pp. 356. Price: 9s. net.

Diary for the Month.

JAN. 21.—Queensland Branch, B.M.A.: Clinical Meeting.
JAN. 22.—Queensland Branch, B.M.A.: Council.
JAN. 25.—Victorian Branch, B.M.A.: Council.
JAN. 27.—Federal Council, B.M.A.: Half-Yearly Meeting (Melbourne).

Medical Appointments.

Dr. W. S. Dawson has been appointed Professor of Psychiatry in the University of Sydney, New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xx-xxii.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Officers.
CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Medical Officers.
FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Resident Junior Medical Officer.
MUNICIPALITY OF GLAMORGAN, SWANSEA, TASMANIA: Medical Officer of Health.
NAMBOUR HOSPITALS BOARD, NAMBOUR, QUEENSLAND: Medical Superintendent.
ZEEHAN AND MONTAGU MEDICAL UNION, ROSEBERY, TASMANIA: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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